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of
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ANNUAL MEETING

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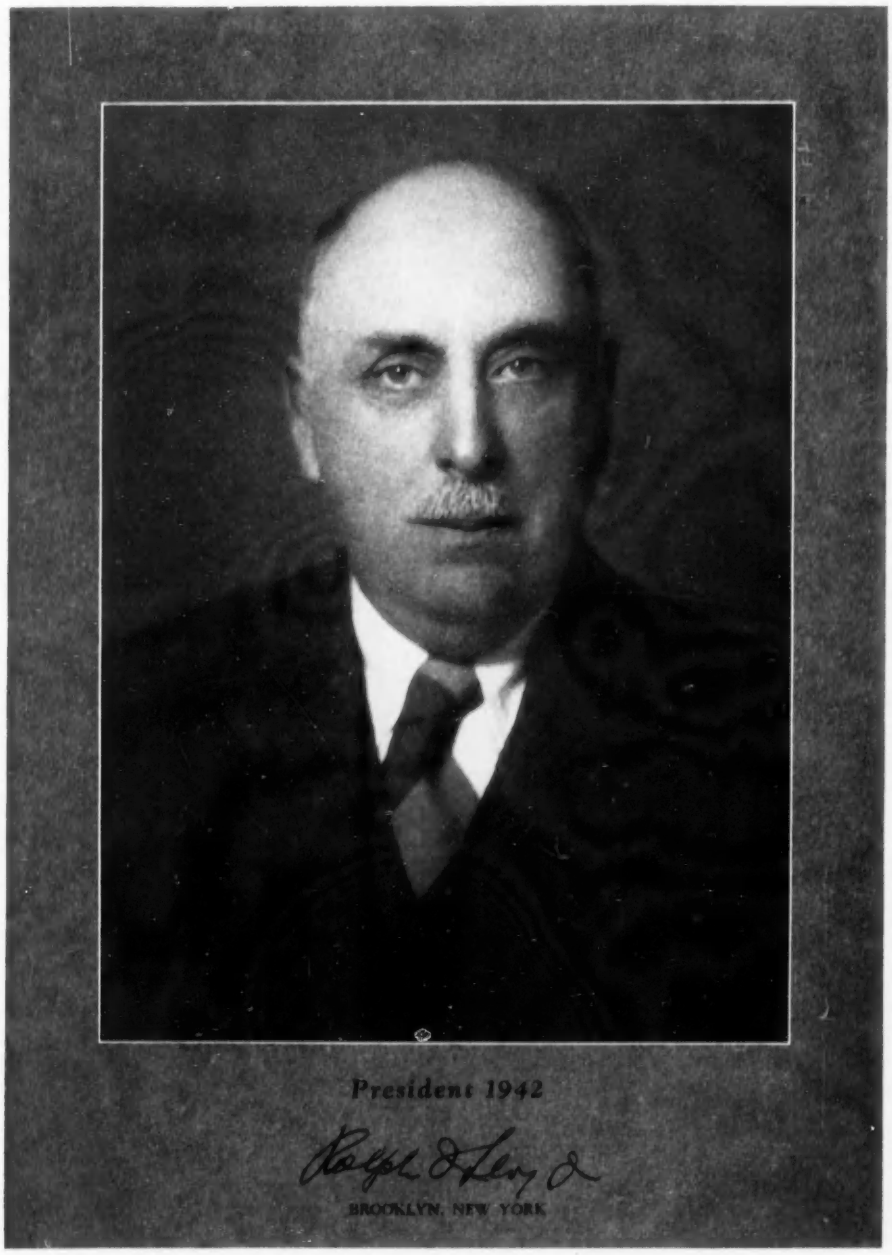

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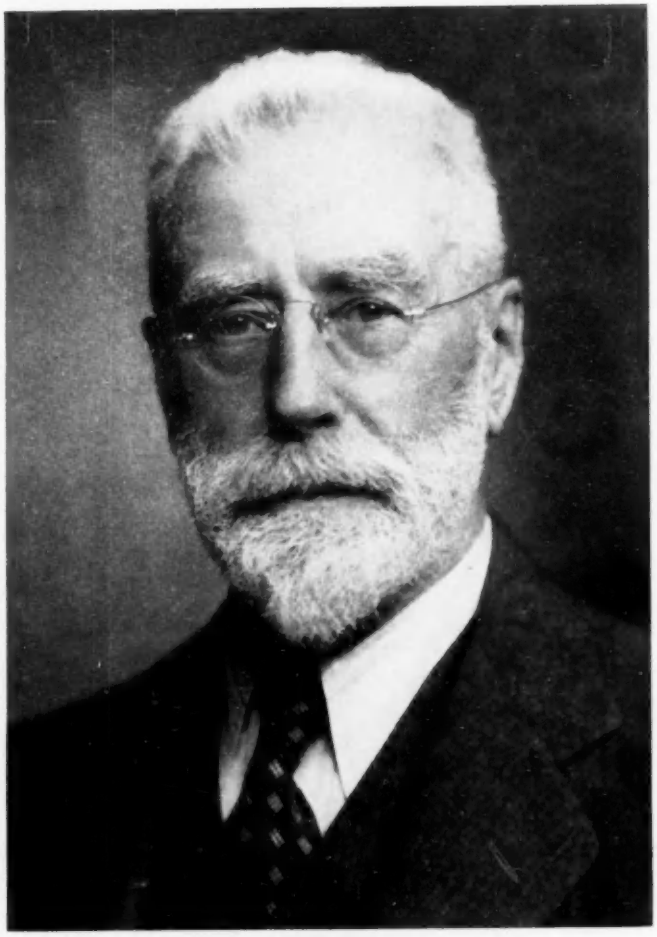

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Address of the President

* * *

EMBRYOLOGICAL BASIS OF EXTRAPAPILLARY COLOBOMA AND CONUS

RALPH I. LLOYD, M.D.
BROOKLYN

THE EARLY embryonic eye has two layers, the inner being the inversion of the anterior segment of the primitive hollow eye bud, from which develops all retinal layers except the pigment layer. The primitive outer layer produces only the pigment layer of the retina. There are no fibers passing from one layer to the other to hold them together. This loose relationship is easily disturbed by fluid accumulating between the two layers of the retina or a shrinking of the contents of the vitreous chamber. This is the anatomical basis of detached retina. In striking contrast is the close relationship at all times between the pigment layer of the retina, the lamina vitrea and the choriocapillaris. This close relationship is plainly seen in sections of the embryonic eye. Furthermore, there is no sharp separation between the choriocapillaris and the deeper layers of the choroid and the adjoining scleral tissue. (Illustration No. 1.) Clinically and with the ophthalmoscope, the pigment layer, the lamina vitrea and the choriocapillaris are in effect a single tissue. This is also true from the pathologist's standpoint. There are two errors of development of the human eye which concern mainly these closely connected tissues; extrapapillary coloboma and the defects located in and about the optic

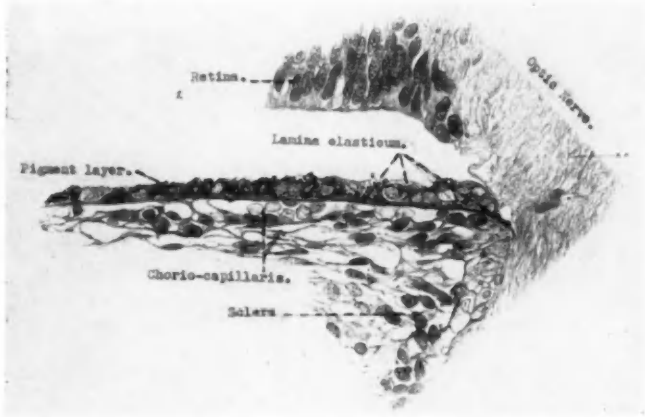

FIG. 1 — (Atlas zur Entwicklungsgeschichte des meschlichen Auges. Bach and Seefelder, 1914.) Section adjoining optic nerve entrance showing the intimate relation of the pigment layer of the retina and the chorio-capillaris. The lack of cohesion between the pigment layer and the remainder of the retina is evident.

Presented at the Forty-Seventh Annual Session, of the American Academy of Ophthalmology and October 11-14, 1942, Chicago, Illinois.

disc and known in its ascending grades as conus, coloboma or ectasia (staphyloma). The number of these lesions examined both with the ophthalmoscope and the microscope is relatively few but the reports agree in essential details.

Extrapapillary coloboma is named to distinguish it from defects due to defective closure of the optic fissure. These characteristic lesions are located in and about the posterior pole, are multiple or single, of variable size and unilateral or bilateral. They are sometimes familial and are of importance because of their effect on vision. I have seen these typical lesions in a childless man and also in one of two children in each of the families of his two brothers. The description (Illustration No. 2) given of the grandfather indicates he must have been similarly affected. Lindsay Johnson described extrapapillary coloboma and conus in 1890. He reported the following characteristics: (1) sharp, clear outlines, (2) pigment anterior to the arteries, (3) lesions unchanged over a long period, (4) floor very white because of the exposed sclera, (5) veins that do not cross the lesion unless it is near the disc, (6) absolute scotoma. Johnson regarded the lesions as similar to moles on the skin. He believed they were due to a defective vascular frame work from which the tissue develops. The finger-like pigment lines so often found in these lesions were regarded by him as remnants of this vascular network. The field defect may not correspond to the expected findings in area or form. With the larger lesions, vitreous floaters, myopia and astigmia are often found. Other malformations connected with the retinal vessels occur. As examples of this last group, retinal veils and folds are most common. Another striking feature is a marked tendency to sclerosis of the choroidal vessels in the affected area even in young persons. If the macular area is involved, the visual acuity, judging by test type standards, is always poor. On the contrary, even if the lesion is bilateral, and though the patient may not be able to read a newspaper, he can get about the streets with little difficulty and may conduct his business with the help of someone to do his reading and writing. The manner of using the eyes is characteristic. (Illustration No. 3.)

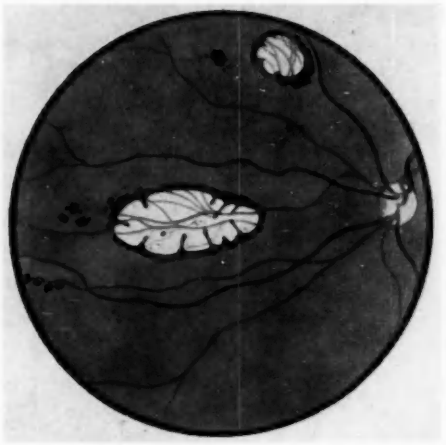

FIG. 2 — **Extrapapillary Coloboma** of Simple Type. Unilateal, no central vision, congenital pigment clumps.

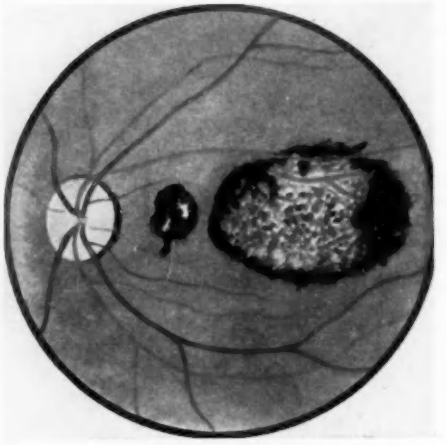

FIG. 3 — Bilateral Extrapapillary Coloboma. Right eye enucleated after injury. Left eye — 10/50 with correction. Patient operates a lathe.

The patient looks out of the corners of the eyes in alternation while moving the head from side to side. Serious involvement of the macula of whatever kind usually produces nystagmus. However, even in the bilateral cases of this type with very large macular lesions, nystagmus has not been seen in the cases coming under my observation. This is in keeping with the observation previously made that the vision is much more useful than would seem possible. Ida Mann placed these lesions under three headings: The first is the pigmented lesion of the choroid with retinal vessels running over it without interruption. This dates from the sixth month of foetal life. The second is a non-pigmented lesion with a pearly white background and ectasia of the sclera. This is classified as a destructive change and dates from the fourth month of foetal life. The third type has abnormal retinal vessels which connect the retinal and embryonic circulation. This lesion appears as early as the third month. Like all exact formulas in medicine, there are exceptions as will be shown. (Illustration No. 4.) Microscopical examinations of these lesions have been made by Treacher Collins, Sorsby and Oliver, Wexler and Last and Rintelen. The group of allied defects about the optic disc have been examined by von Szily and Elschnig and their findings agree in the essential details. Only the inner retinal layers are found in the affected area. The pigment layer is absent and

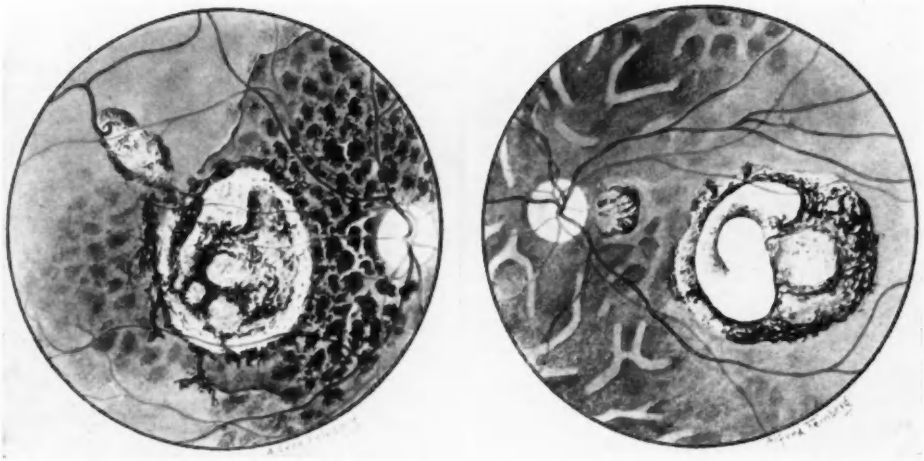

FIG. 4 — Bilateral Extrapapillary Colobomas. 4/200 and 3/200. Patient cannot read the paper but conducts a business and goes about alone. The three features of these defects assigned by Ida Mann are seen here in each eye. Exposure of sclera with pigment-ectasia of the sclera-communication of retinal and deep choroidal circulation.

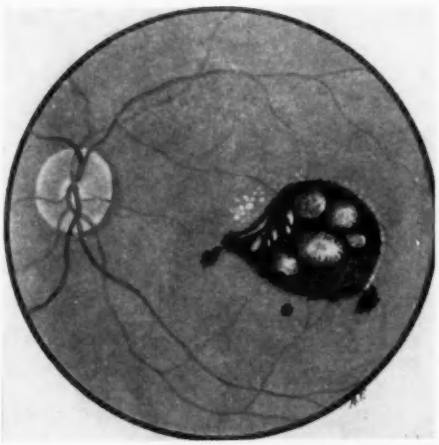

FIG. 5 — Unilateral Extrapapillary Coloboma. No central vision.

the choroid at most is only a few shreds of pigmented tissue. The sclera may be reduced to a thin layer which may bulge forth producing the change in level seen in the more serious defects. There are no signs of inflammation. (Illustration No. 5.) The consensus of opinion is that the original lesion is a defect of the pigment layer of the retina. The choriocapillaris does not develop in the absence of this layer. This in turn affects the sclera and inner layers of the retina. Mann regards the lesions as due to intra-uterine inflammation, but the bilaterality, familial tendency and a connection of retinal vessels with those of the deepest choroidal level denies this assumption. (Illustration No. 6.) The differential diagnosis is of importance in this day of compensation and life insurance with their clauses including liability for loss of vision. The lesions of extrapapillary coloboma and conus must be distinguished from the healed changes of chorioretinal tuberculosis, a type of hereditary macular degeneration with

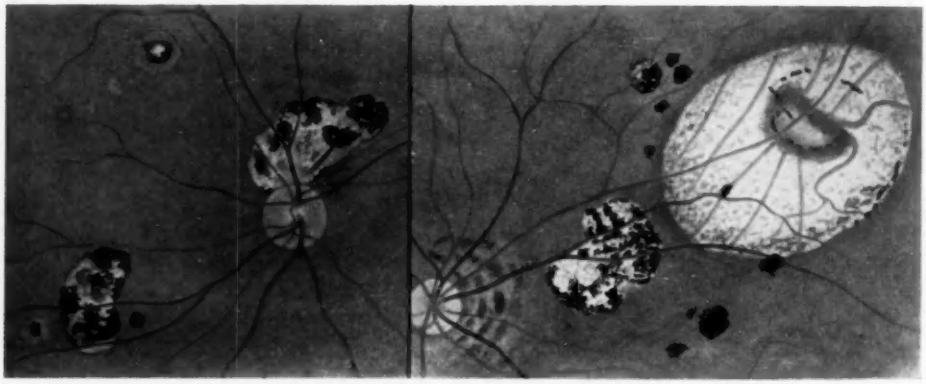

FIG. 6 — Bilateral Extrapapillary Coloboma. V. c.c. 20/20 and 20/33.

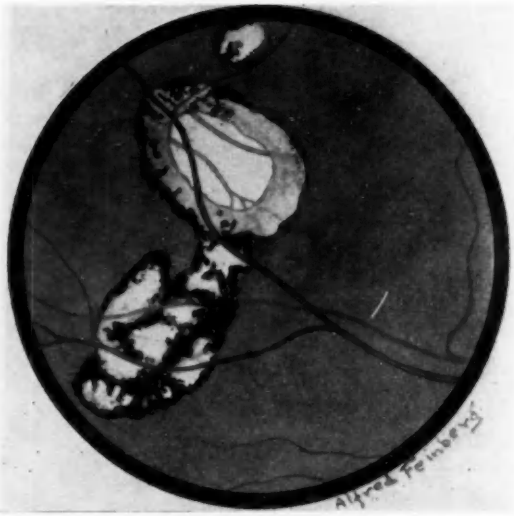

FIG. 7 — Healed Tuberculous Chorioretinitis. This lesion is a part of the defect produced by five attacks.

pigment masses, and the results of trauma with hemorrhage. I do not believe healed tuberculous lesions can always be definitely differentiated. Tuberculous chorioretinitis is not often bilateral, the pigment border with fingers of pigment running into the defect is not found, the vitreous may show anything from a dense haze to retinitis proliferans, there is no ectasia of the sclera, and no communication of the two vessel systems. A reliable history is of use. In many cases of tuberculosis the slit lamp will show bits or even clumps of fibrin on the posterior surface of the cornea, nodules on the iris margin, or synechia. Atrophy of the iris and involvement to a lesser degree of the second eye may also be found. (Illustrations No. 7 and 8.) Hereditary macular degeneration is always bilateral, and the type with marked pigmentation shows no finger-like processes, the sclera is not widely exposed, nor are the vessel connections found. These patients are unable to work for a long time before their eyes reach the stage in which the ophthalmoscopic appearance is at all confusing. Other cases in the family should be sought. There is little likelihood of confusing a tuberculous lesion with healed retinal hemorrhage due to trauma unless it is a unilateral change. Birth injuries with intraocular damage may possibly confuse the ophthalmologist in making a diagnosis in rare cases. (Illustrations No. 9 and 10.) The defects located about the optic nerve entrance known as conus, coloboma or ectasia (staphyloma) have the same defective development of the pigment layer of the retina and the choriocapillaris. In addition, there may be oblique insertion of the optic nerve, defective closure of the optic fissure at the junction of the nerve and globe, defective insertion of the lamina cribosa, and pockets at the side of the nerve head. We will confine our discussion to the lesions at the side of the nerve head. If there is no difference in the level of the floor of the defect and the normal fundus, it is known as conus.

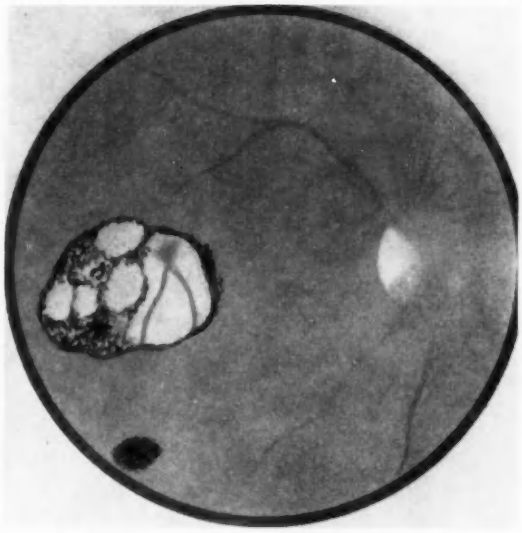

FIG. 8.—Healed Tubercular Chorioretinitis under observation 14 years. This iris is fading, fibrin deposits on the posterior corneal surface are always present and there are crops of nodules on the margin of the pupil.

The name coloboma signifies a difference in level while the term ectasia or staphyloma describes the advanced change involving the sclera with local bulgings. (Illustration No. 11.) These lesions are all congenital and stationary and are not to be confused with the acquired crescents of myopia. Vossius found the location of 111 defects of the congenital form as follows: Below — 67 per cent, down and in — 7 per cent, in — 8 per cent, up—4.5 per cent, up and out—7 per cent, down and out — 5 per cent. Predominance of the defect below the disc is expected, as that is a critical point at the junction of the fissure in the optic nerve and the fissure in the globe. It is the last point of closure of the optic fissure and structurally the weakest point. In contrast with these figures are those of the acquired crescents of myopia: Below—6 per cent, out—79 per cent, annular—11 per cent, up—4 per cent. The typical conus is a crescent shaped section adjoining the disc, and is of lighter color than the normal fundus because the pigment layer of the retina and its associated layers are lacking. The optic nerve is usually inserted at an oblique angle so there is an overhang of choroid on the side of the disc opposite the conus, giving the impression of a shortened diameter. In the more pronounced type, in addition to the absence of the pigment layer and choroid, the sclera is thin and stretches so the floor is plainly on a lower level than the adjoining fundus. (Illustration No. 12.) At birth, the exaggerated type is very similar to the fundus of high myopia in adults. The optic axis is much longer than normal, the temporal segment of the tapetum is thin, its deeper vessels visible and there is marked difference in the refraction of the macular area and that of the disc, sometimes as much as 12 diopters. The

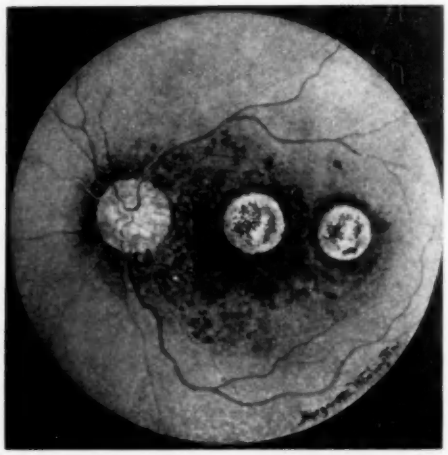

FIG. 9 — Extrapapillary Coloboma (unilateral). Burton Chance. Ophthalmic Record XXII, p. 1, 1913. V. = Fingers at three feet.

FIG. 10 — Symmetric Macular Degeneration in a Brother and Sister. Burton Chance. American Journal of Ophthalmology, III, p. 241, 1920. V. with correction 5/50-o.u. Colo. blind and contracted fields. Failing vision began at 10 years of age.

eye is often myopic, astigmatic and amblyopic. A depressed conus is often associated with a coloboma of the nerve, in the floor of which may be a depression which may communicate with a pocket at the side of the nerve sheath. Several colobomas have been examined with the ophthalmoscope and also with the microscope, and the basic errors of extrapapillary coloboma and the various types of conus agree. Microscopically, the main feature is a failure of the pigment layer to reach the optic nerve entrance. The choriocapillaris does not develop and the sclera, the deeper layers of the choroid and the inner layers of the retina suffer in consequence. The lamina cribrosa develops late in embryonic life from the choroid near the scleral ring. If the pigment layer fails,

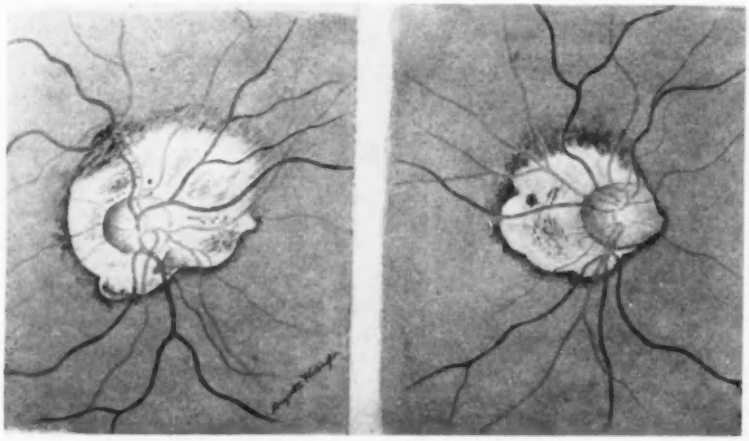

FIG. 11 — Bilateral Nasal Conus. Burton Chance. — 5.00 — 3.00 ax. 105° — 6/9. Male 60 years. — 4.00 — 2.00 ax. 75° — 6/9. Difference in level — 4 Diopters.

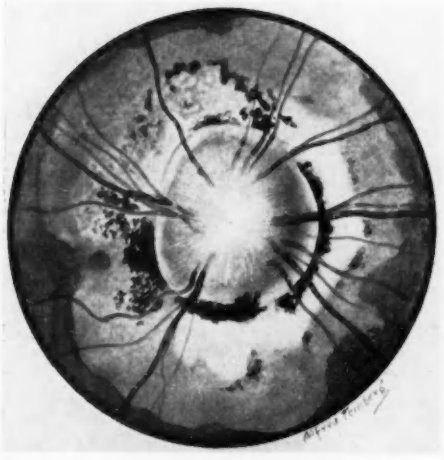

FIG. 12 — Large Conus Annularis, with myopia, astigmia and amblyopia. Unilateral. Dr. Harold Lowenstein.

there is often a space at the disc margin where the lamina cannot attach; it may drop back and attach, or a pocket may develop beside the nerve like a hernia suggesting increased intraocular pressure at some time during the developmental stage. Cross sections of the typical myopic eye including the disc and its neighboring structures are very similar to sections of pronounced conus and coloboma. This is shown as the two are compared. (Illustrations 13 and 14.) Both are familial, but one is congenital while the other is acquired. The congenital lesion may be the basis of advanced myopic changes at an early age. Illustrations of sections of classical cases examined before and after removal are offered to clarify in order to save print-

ing and to ease the strain upon my auditors. A discussion of conus immediately becomes one of coloboma of the optic nerve, pits in the nerve head and cysts of the optic nerve sheath, inasmuch as the conus is the initial stage of a process that may go on to involve other closely related tissues.

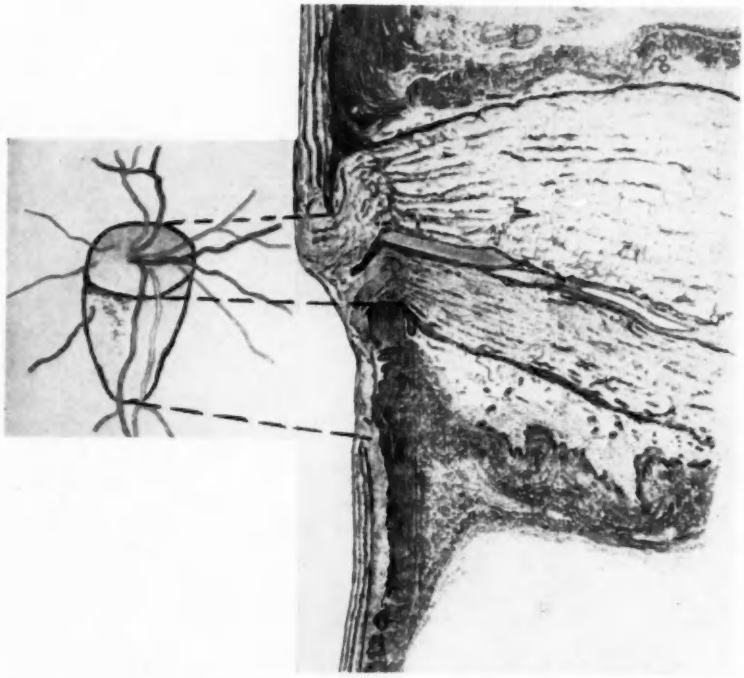

FIG. 13 — (Elschnig. Das Colobome am Sehnerveneintritte und der Conus nach unten. Archiv fur Ophthal., LI, p. 391, 1900.) Sketch of compound myopic astigmic eye with micro section of the same eye placed to show the ophthalmoscopic appearance of the chorioretinal defect.

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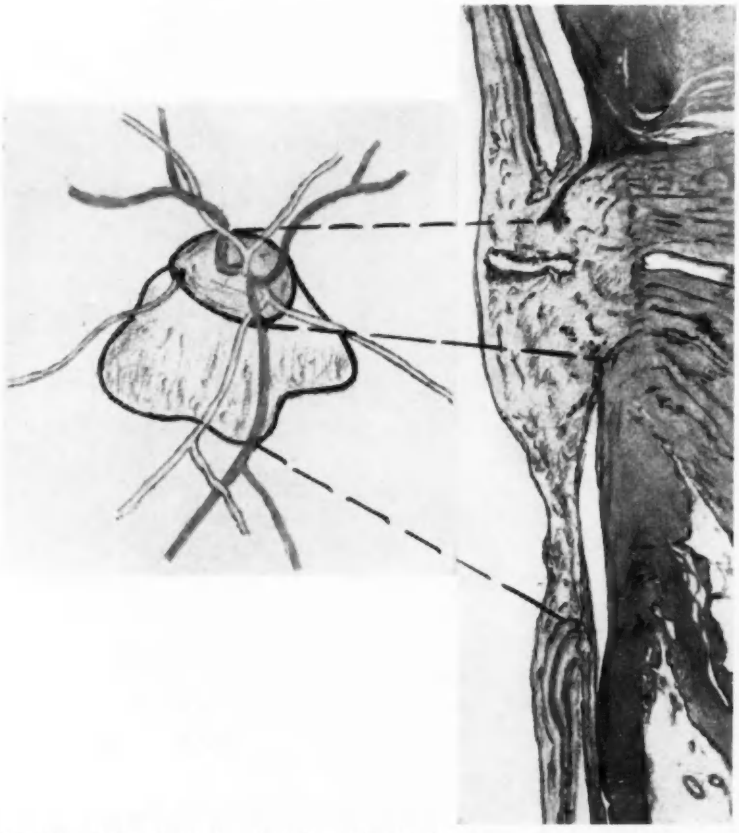

FIG. 14 — (Elschnig. Weitere Mitteilung über das Colobom am Sehnerveneintritte und den Conus nach unten. Archiv für Ophthal., LVI, p. 49, 1903.) Sketch of an eye with "Conus nach unten" and micro section of same eye showing chorioretinal defect.

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Address of the First Vice President

* * *

THE ROLE OF THE OTOLARYNGOLOGIST OF TODAY — 1942

WALTER H. THEOBALD, M.D.
CHICAGO

WHEN a large group of specialists comes from far and near to a scientific session of a medical organization such as this, the occasion is a significant one. Significant because we are not unmindful of the sacrifices many of you have made in attending this gathering and the sacrifices made by those who are serving in the armed forces. Significant, furthermore, because of the numerous problems before us, problems which this Academy must help solve, problems which in some manner or other affect each and every one of us and which may influence not only our present lives but our future. War demands such sacrifices.

You are here because you are interested in the solution of these problems. That is the democratic way of life. It is the method to which we have become accustomed, because, in this country men whose voices are heard and whose sentiments can be freely expressed are really men. You gentlemen who comprise this Academy have a voice in its conduct because its government is of the representative form, the type of government which has so rapidly developed this country into the greatest nation on earth. A bored nation is impossible to govern but a nation occupied by work in which it believes is a useful nation and also a happy one. God bless work!

You, who have gathered here, have done so for a purpose. You desire to keep abreast of the times in your own fields. You desire to exchange ideas and experiences with your colleagues, and you desire to learn and familiarize yourselves with all that is new and worth while in our specialty. By reason of your efforts in research and carefully recorded clinical observations there has been definite scientific progress in otolaryngology. You are the men who are responsible for this progress.

When one attempts to evaluate the progress made with chemotherapy in otolaryngology, he is bewildered. He looks back with regret to recount the number of lives that might have been saved with the newer sulfonamide drugs, had they been available a few years ago.

When one reviews the more recent therapeutic successes in certain rhinological affections by the newer developments in allergy, one appreciates that our present knowledge of this subject represents a definite and decided advance. Likewise, the achievements in the otological field have been very noteworthy because constructive, rather than destructive, procedures have been followed. By relentless work along these lines our accomplishments have kept pace with and, in some cases, outdistanced the marks set by some of the other special fields in medicine. As André Maurois says in his *Art of Living*, "The average length of a man's life one hundred years ago was about forty years. Today, in the most highly civilized countries it is nearly sixty — a rapid change and one which allows us to think that, if

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wars and revolutions do not impede the progress of hygiene, a hundred years may be the normal length of life in the next century."

But now we are living in perilous times, under difficult conditions, and with the added tension of uncertainty. Today we are here, next week or next month many of us may be in remote parts, away from civilian practice, compelled to adjust ourselves to new environments. The old adage that the doctor was the servant of the people is truer today than ever before. He is the servant of humanity, always ready to play his part. Need I detail to you the place of the American doctor in the world struggle? Need I recall to you the present urgent responsibility of the medical profession? Need I relate facts concerning the greater efforts which will be required of those physicians who remain to care for civilian needs? I doubt whether I can add anything to the information you already have. But I am confident that the time is now at hand when much more serious consideration should and must be given to the part you and I are to play in this great world conflict. Barbarous as wars are, they too contribute to medical science. The rapid development of aviation medicine illustrates this statement. Aviation medicine embraces new and important phases of ophthalmology and otolaryngology. Therefore, our specialties must be adequately represented to handle these newer problems and they must keep abreast of all the newest developments which are presented by a vastly increasing branch of military service.

And when the struggle is over, our work will just begin. Those who have passed through the reconstruction period of the last war have at least an idea of what will be expected of us. The work of rehabilitating the maimed and the injured will be overwhelming. Sir Harold Gillies, who was the honored guest of this Academy one year ago, brought this fact vividly to our attention. We must be equal to the task. Great progress has already been made in reconstructive surgery in the field of otolaryngology, and even greater progress and results are to be expected and hoped for through our earnest efforts. It takes such a crisis to bring to a vivid realization the fact that no specialty can survive if it fails to meet the increasing demands made upon it.

It is regrettable that the trend of the times renders a review such as this essential for a group like ours. You will agree, however, that these are the facts, and we must be ready and willing at all times to give our services to the call of humanity. War imposes these tasks upon us. Your interest in the progress of our specialty, your cooperation in, and contribution to, our war efforts and your loyalty and faith in maintaining principles for the maintenance of our democratic way of life will, I am sure, give added confidence to you who are entrusted with these tremendous responsibilities.

I am sure that I voice the will and desire of our Academy, and of everyone of you here, to stand by devotedly to our task so that civilization may be saved, so that scientific medicine may continue to develop, and so that we, in our representative specialties, may in a small way contribute to this progress, for the betterment of all mankind.

Symposium on Chemotherapy

CHEMOTHERAPY IN OPHTHALMOLOGY

JOHN G. BELLOWS, PH.D., M.D.
CHICAGO

IN THE last few years new chemotherapeutic agents have made their appearance. These agents consist of the well known sulfonamide group, already proved valuable clinically, and lesser known substances derived from molds, fungi and bacilli, i.e., penicillin from penicillium, tyrothricin from soil bacteria, actinomycosin from actinomycosis, etc. The ophthalmological literature contains practically no reference to the latter substances. Fleming[1] states that penicillin is approximately four times as potent as sulfathiazole, twenty times as potent as sulfapyridine and 100 times as potent as sulfanilamide. It has an important advantage over sulfanilamide in that it is not inhibited by secretions, pus and large numbers of organisms. Tyrothricin consists of 15 per cent gramicidin and 85 per cent tyrocidin, both of which show marked bactericidal powers for gram positive organisms. Gramicidin, about 25 to 50 times more potent than tyrocidin, is effective in amounts as low as 1 gamma per billion organisms.[2] In the absence of peptones and similar inhibitors, tyrocidin is effective not only against gram positive but also against gram negative organisms such as the meningococcus. Penicillin, gramicidin and tyrocidin actually kill organisms, whereas sulfonamides are merely bacteriostatic.[3] Thus, if the former chemotherapeutic agents should prove to be clinically applicable they would be superior in infections of the cornea, vitreous and other avascular areas to sulfonamides which depend upon the mobilization of leucocytes for the destruction of the invading organisms.

In the future, it is possible that certain ocular infections may be found more responsive to these substances than to the sulfonamides. Further, when an organism has become sulfonamide-fast these might be substituted. It is possible that the already marked effectiveness of these chemotherapeutic agents may be increased by combining them with other substances, as has been done with the sulfonamides. Branham,[4] working with anti-meningococcic serum and sulfonamides, and other investigators[5] using anti-pneumococcic serum found the combination more effective than either substance alone. Zaytzeff-Jern, et al.,[6] reported that the action of sulfonamides on staphylococcic infections is enhanced by the addition of bacteriophage, while Thygeson[7] and Allen[8] obtained similar results with sulfonamides plus staphylococcic toxoid. The following writers found an increased anti-bacterial effect when other agents were used with sulfonamides: Osgood, et al.,[9] adding neoarsphenamine, Neter[5] adding azochloramide and Goldberger[10] using zinc peroxide, iodine and potassium permanganate.

The author[11] was unable to obtain penicillin but tried tyrothricin* experimentally and found that when placed in

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* Furnished through the courtesy of Merck and Co.

the conjunctival sac in one-half per cent suspension it is non-irritating but when used in high concentration it produces considerable irritation and even cloudiness of the cornea. Clinically it was applied in several cases of acute conjunctivitis with unimpressive results. However, further investigation from the ophthalmological standpoint seems to be warranted. In this report, the subject matter is limited to the sulfonamide drugs and of this group mainly to sulfanilamide, sulfathiazole, sulfapyridine and sulfadiazine.

Kolmer and Tuft[12] write, "Until the discovery of the effectiveness of the original prontosil in the treatment of streptococcal and staphylococcal infection, the chemotherapy of systemic bacterial diseases was largely disappointing." Likewise the use of sulfonamides, passing within a short time from the purely experimental stage to a definite clinical acceptance, has revolutionized the treatment of many ocular infections. The selection of the drug, whenever possible, should be based upon a precise knowledge of the bacteria involved and the penetrability of that particular drug into the ocular tissues. Sulfonamides exert their maximum therapeutic effect only with satisfactory concentrations at the site of the infection. For most infections it has been found that blood and tissue concentrations between 5 and 10 mg. per cent are usually adequate to ensure chemotherapeutic effectiveness; for less susceptible organisms, e.g., staphylococcus, much higher concentrations of the drugs are required. Large dosages for short intervals are preferable to small dosages for long periods for there is evidence that inadequate concentrations of the drugs may not only be ineffective in combatting infections but may actually produce a state of hypersensitization which precludes the use of sulfonamides, or a sulfonamide-fast organism which does not respond even to larger dosages if the need for such treatment should arise.[13] For example, if a simple corneal ulcer, treated with subminimal dosage, develops into a serpent ulcer and then into an endophthalmitis, the seemingly logical treatment would be greatly increased dosage, which, however, in the first instance (hypersensitization) would not be tolerated, and in the second instance (sulfonamide-fast organism) would be entirely ineffective. It is obviously important, therefore, to acquire a knowledge of the penetration and distribution of the sulfonamide drugs after oral and local administration.

OCULAR PENETRATION OF SULFONAMIDES AFTER ORAL ADMINISTRATION

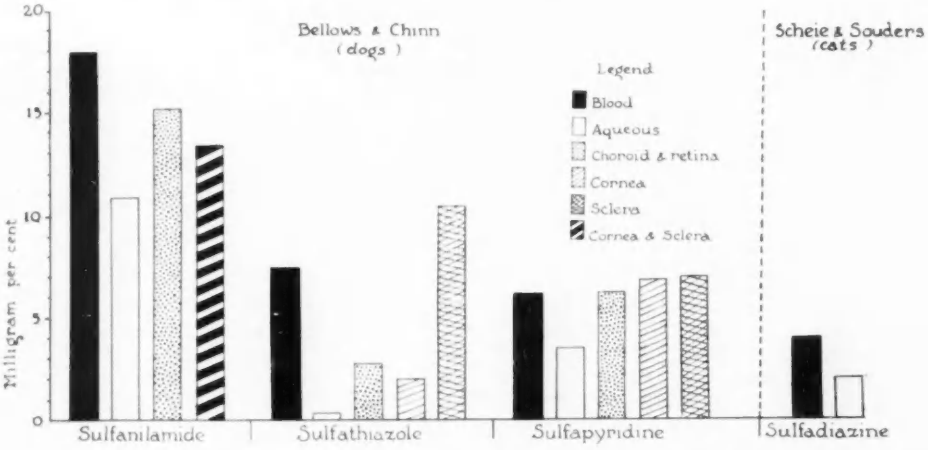
Investigation of the four most commonly used sulfonamides — sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine — reveals that sulfanilamide yields the highest concentration in the blood and ocular tissues and fluids. Much lower concentrations are reached by the less soluble drugs, sulfathiazole and sulfadiazine; whereas the value for sulfapyridine lies between the two extremes.[14 a,b,c,d,e,f,g] In fact, the concentrations of sulfathiazole and sulfadiazine in the eyeball are, with the exception of those in the cornea and sclera, below the value accepted for chemotherapeutic effectiveness. (Fig. 1.) The consistently high concentrations of the sulfonamides in the cornea noted by Bellows and Chinn and confirmed by Meyer, Block, and Chamberlain[15] are explained by the latter investigators as a process of adsorption, which in turn may account for the remarkable curative results obtained in trachomatous lesions of the cornea.

Therefore, it may be stated that under ordinary conditions it is only with sulfanilamide or sulfapyridine given orally that one is reasonably certain of providing adequate concentrations in the ocular tissues and fluids. However, it must not be concluded that sulfathiazole

and sulfadiazine are contraindicated in intraocular infections for it has been demonstrated that the concentration of these drugs in the eyeball may be increased by the application of heat or mecholyl, or by paracentesis, or by inflamation resulting from infection.[14 a,e]

OCULAR PENETRATION OF SULFONAMIDES AFTER LOCAL ADMINISTRATION

There are two obvious advantages of local application: first, a high concentration is rapidly achieved; second, systemic toxic manifestations are avoided. fectiveness. The local application of sulfapyridine, sulfathiazole and sulfadiazine produces concentrations in the aqueous humor less than half the required strength. The penetrabilities of the drugs investigated, in descending order, are: sulfanilamide, sulfapyridine, sulfadiazine and sulfathiazole. The penetrability varies not only with the drug used but also with the animal, that through the cornea of the rabbit being far greater than that through the cornea of the dog.[14 d] Judging from the findings of Lou and Pan the penetration



Concentrations of Sulfonamides in the eye following a single large dose (oral).

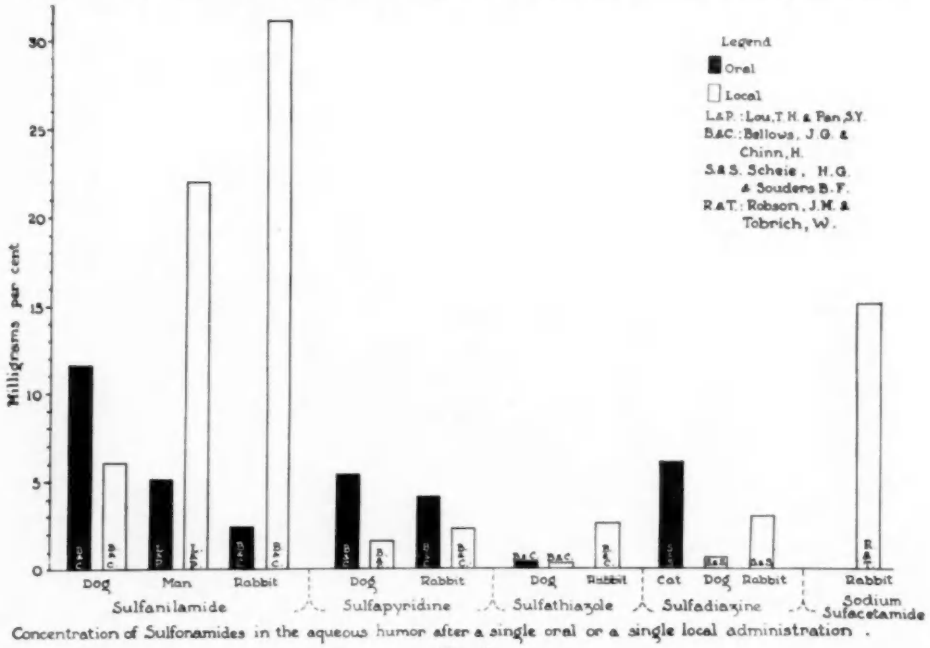
FIG. 1

As soon as the therapeutic value of sulfonamides in ocular infections became evident, investigators[14 a,d 16,17,18,19] sought to determine whether the amount reaching the ocular fluids and tissues following topical administration would be equal to that in oral administration. Their results revealed that, following a single dosage, all the drugs with the exception of sulfanilamide reached a higher concentration by oral than by local administration. Due partly to its greater solubility and small molecular size, sulfanilamide when applied topically reaches a concentration in the eye more than ample for chemotherapeutic effect. through the human cornea approaches that in the dog (Fig. 2).

Recently Boyd,[20] using sulfathiazole, and von Sallman,[21] using sulfadiazine, have demonstrated that a marked increase in the concentration of these drugs in the cornea and aqueous humor of the rabbit's eye may be achieved by means of iontophoresis. Using a low current no untoward effects were observed. By this method apparently one may attain within the eyeball an adequate concentration of even those sulfonamides with low powers of penetration.

At the present time the author has under investigation a simple and yet effective method of increasing the penetrability of sulfonamides through the use of agents which lower surface tension. Some organic compounds such as bile salts, aerosol, sodium laurelate, zephiran, duponol, ocenol, etc., by reducing the surface tension facilitate penetration through the cornea. For example, the greatest concentration of sulfathiazole within the aqueous humor following local application is less than 1 mg. per cent; with a wetting agent the concentration reaches 10 or even 20 mg. per cent. Further studies are under way to determine which of these substances are most practicable for ophthalmological purposes, since some of them in high concentrations produce cloudiness of the cornea. Although these investigations are still in the incipient stage, it seems likely that we may have at hand a substitute for the oral treatment upon which we now rely in certain ocular infections.

Our investigation of the distribution and penetration of the sulfonamides leads to the following practical conclusions: for intraocular infections sulfanilamide and sulfapyridine given orally, or sulfanilamide used topically, or any of the four drugs employed with iontophoresis or with a wetting agent will provide an adequate concentration in the ocular tissues and fluids; for conjunctival and corneal disease any of the four drugs in powder or ointment* form will give an adequate concentration. At this point it is pertinent to mention a misconception of some authors who report the local use of neoprontosil. Since neoprontosil is reduced to sulfanilamide only within the body[22] it is obvious that any apparent local effects cannot be ascribed to its sulfanilamide action and its effectiveness is no greater than that of any non-irritating "drops."



Concentration of Sulfonamides in the aqueous humor after a single oral or a single local administration.

FIG. 2

* Ointment prepared according to Guyton's recommendation for No. 8 formula.

OCULAR MANIFESTATIONS OF SYSTEMIC SULFONAMIDE TOXICITY

General systemic toxic manifestations are not under consideration, but the report of Martin, Fisher and Thompson[23] is noteworthy in that they demonstrated that the addition of calcium glucuronate, cysteine, ascorbic acid and amino-acetic acid diminishes toxicity without reducing the efficacy of the sulfonamides.

The first report of an ocular manifestation of sulfonamide intoxication was given by Bucy,[24] who described an optic neuritis which subsided upon withdrawal of the drug. A second case was mentioned by Duggan.[25] Baker[26] described an individual who upon receiving 370 grains of sulfanilamide in 4 days developed round hemorrhages along the course of swollen arterioles. Ocular manifestations vaguely termed "temporary dimness of vision" and "temporary blindness" have been reported in the literature by Colebrook and Purdie,[27] and Bloom, Leech and Shaw.[28] Rosenthal[29] observed field changes by means of angioscotometry. Sulfathiazole has been shown by Haviland and Long[30] to produce a conjunctival and scleral injection, restricted to the exposed portion, accompanied by considerable burning and watering. Like other drugs, which in large quantities act as poisons, sulfonamides may produce in the sclera a yellowish discoloration indicating hemolytic or hepatic jaundice, and in the conjunctiva a bluish tinge or pallor suggesting cyanosis or anemia. Transient myopia[31-42] is by far the most frequent ocular disturbance described. A return to the normal refractive state rapidly follows withdrawal of the drug. In many of the cases when a cycloplegia was applied the degree of myopia was unaffected, thereby indicating that the changes producing the myopia lie in the crystalline lens rather than in the ciliary body. Bellows and Chinn have demonstrated that sulfanilamide permeates the lens, reaches its peak concentration there and leaves more slowly than in other ocular tissues and fluids. This condition is analogous to that found in diabetes mellitus which results in a "dry salt retention" with its accompanying refractive changes.

LOCAL TOXIC EFFECTS OF SULFONAMIDE DRUGS

While the toxic manifestations following oral administration have been widely reported and are well known, the local toxic effects of sulfonamides on the ocular tissues have received practically no attention. Bellows and Chinn state that the local application of sulfanilamide powder to the normal eye for 2 hours results in a moderate reaction consisting of chemosis and staining of the corneal epithelium. Upon discontinuance of the drug the reaction subsides within a short time. Recent work by the author[43] indicates that in the absence of normal epithelium, sulfonamides cause a severe injury to the cornea. Thus, if the corneal epithelium of rabbits is removed, the healing time in the untreated eyes averages 5.1 days, while in the sulfonamide-treated eyes the average is 13.3 days. The time of regeneration in the latter group would have been greater had it not been that in some instances the application of the drug was stopped at the time of complete recovery of the controls; in other instances the epithelial regeneration of the cornea did not progress beyond a few millimeters in the periphery until the cessation of drug administration. Sulfonamides not only cause a delay in the healing of corneal epithelium but cause scars which are highly vascularized. Therefore, after a laceration or abrasion when a minimal amount of scarring is desirable, as on the face and cornea, the local use of the sulfonamides should be limited to cases in which dangers of infection outweigh the cosmetic factor.

INDICATIONS FOR ORAL AND LOCAL ADMINISTRATION

Because of the toxicity of sulfonamides, oral administration should not be utilized indiscriminately or for every slight infection, particularly those infections which are self-limited or easily controlled, but should be reserved for serious conditions.

When infection has begun in the interior of the eyeball or has progressed beyond the reach of topical application, oral administration seems to be indicated. The apparent corollary would be that local applications are indicated for superficial infections. This deduction, however, is not entirely true. For example, in conditions such as trachoma or gonorrheal conjunctivitis, where local therapy might be expected to yield most effective results, clinical experience proves oral therapy to be the superior method. Finally it must be remembered that a combination of both oral and local administration may in some instances be the best form of therapy.

The effectiveness of local sulfonamide application to the eye is governed by certain local conditions. Since the cornea is avascular and since the drugs are bacteriostatic rather than bactericidal, the effect is less marked in this tissue than in a vascularized tissue or in the bloodstream with its full complement of bacteria-destroying leucocytes. Further, the effectiveness in this region is decreased by the secretion and detritus so often associated with ocular disease and the frequent use of local anesthetics such as novocaine and related substances whose structural formulae resemble that of para-aminobenzoic acid. The presence of secretion itself in gonorrheal and trachomatous infections, and all these influences in infectious corneal ulcers, may account for some of the failures in ocular therapy. Although our present knowledge is inadequate to give absolute indications for local and oral administration of sulfonamides in many ocular infections, a working plan is essential. In the following conditions recommendations of the majority of ophthalmologists for the treatment of ocular infections are presented.

ERYSIPELAS

Erysipelas, although not primarily an ocular condition, is of interest to the ophthalmologist because of the site of predilection — the inner canthus of the eye — a factor leading to ocular complications: lid abscess, corneal ulceration and orbital cellulitis. The disease is caused by the hemolytic streptococcus. Among the first to use sulfonamides in the therapy of erisypelas were Meyer-Heine, and Huguanin.[44] These authors reported that in 150 cases treated orally with a sulfonamide preparation, the fever and the lesion subsided within 48 hours. Similarly excellent results have been reported by British and American writers, who stress a marked decrease in mortality.[45,46,47,48,49] The keynote of success in the treatment of erysipelas is the early administration of sulfonamides as shown by Rantz and Keefer,[50] who state that it is impossible to demonstrate any effect of sulfonamides on the course of the disease if given after the third day. Shank, et al.,[51] state that "The prompt response of erysipelas to sulfonamides makes this the treatment of choice and renders other therapeutic measures obsolete."

PYOGENIC INFECTION OF SKIN OF THE EYELIDS

Impetigo contagiosa is the most common of all the pyogenic infections of the skin and at times involves the skin of the eyelids. It may be of streptococcic, staphylococcic or mixed bacterial origin.[52,53,54,55,56] It responds to the use of a 10 per cent sulfanilamide ointment within 4 to 7 days, according to Combes-Canizares.[57] However, Schlesinger and Martin[58] believe that a combination of oral and local administration of sulfo-

namides is superior to either one alone. Likewise, in furunculosis and eczematoid lesions from which large numbers of staphylococci may be recovered, sulfonamides are of value. In addition, it has been shown that other dermatologic diseases caused by pyogenic organisms, such as superficial ecthyma or secondary infections of pre-existent dermatoses, clear more rapidly with sulfonamides than with any other form of treatment.[59-60]

MOLLUSCUM CONTAGIOSUM

Molluscum contagiosum is a contagious virus disease sometimes involving the eyelids, conjunctiva and cornea. Sommerville[61] reported clinical cures in 6 out of 8 cases treated with sulfapyridine. In the other 2 patients the drug was discontinued because of toxic manifestations. Hill and Downing[62] confirmed this observation and reported a cure in an adult after using 1 tablet of sulfapyridine 4 times daily for 1 week. If these observations are substantiated, there will then be 5 virus diseases known definitely to respond to sulfonamides: trachoma, inclusion blenorrhea, lymphogranuloma venereum, psittacosis, and molluscum contagiosum.

GONORRHEAL OPHTHALMIA

Since the introduction of Credé's method of prophylaxis more than half a century ago, there has been no greater addition to the therapeutic armamentarium for combatting gonorrheal ophthalmia than the sulfonamides. The majority of ophthalmologists are agreed that uniformly good results are obtained in the treatment of gonorrheal conjunctivitis with sulfonamides, in that the period of morbidity, hospitalization and incidence of corneal complications are greatly decreased. Gifford[63] writes that "Sulfonamides in sufficient dosage will, in most cases of gonorrheal conjunctivitis, free the secretion of gonococci more quickly than the use of irrigations and foreign protein alone."

The initial reports of the pioneers Hageman,[64] Levy,[65] Fernandez and Fernandez,[66] and Willis[67] showed the therapeutic value of sulfanilamide. Barbour and Towsley[68] reviewed the reports in the literature and the results of their own cases and concluded that the drug reduces the duration of the disease from weeks to days. In 15 of their own cases, smears became negative in an average of 4.8 days. The report of Bower and Frank[69] soon followed. They secured even more striking results in 15 cases, in that the average number of days necessary to obtain negative smears was reduced to 2.0. Sweet[70] demonstrated, in a review of 100 cases treated with sulfanilamide and in 78 cases treated with sulfapyridine gathered from the literature and his own observations, that only 4 instances of corneal complications occurred in the former and none in the latter group.

More recent writers believe that sulfapyridine and sulfathiazole are superior to sulfanilamide in combatting gonococcal infection. The wide acceptance and usage of sulfapyridine on the continent and by several American investigators attest to its efficacy.[70-81] Most American investigators,[82-88] however, agree that sulfathiazole surpasses even sulfapyridine as an antigonococcal preparation. Its superiority was demonstrated experimentally by Lawrence,[87] who found that sulfathiazole had greater bacteriostatic power than either sulfanilamide or sulfapyridine. Clinical confirmation was offered by Wong,[88] who observed that negative smears are obtained with sulfathiazole in 1 day as compared to the 2 days required for sulfapyridine. As can be seen from the accompanying table presenting 253 cases collected from the literature, the average number of days required to obtain negative smears after using sulfanilamide was 5.8 in 150 patients; after using sulfapyridine it was 2.6 in 85 patients; after using sulfathiazole it was 2.3 in 18

patients. The drug dosage must be adequate from the beginning of treatment or a refractory condition may arise. Such refractoriness, as has been shown for pneumococcus, may be due to an acquired tolerance, which may be developed in vitro by a low concentration of the drug and in vivo by administration of inadequate dosages. The need for intensive treatment is emphasized by Willis,[67] Guyton,[18] Jasseron and Morard,[89] and others. Barbour and Towsley[68] point out that newborn infants tolerate sulfonamides well and require larger quantities in proportion to weight than adults to obtain a blood concentration of the desired level. A blood sulfonamide concentration between 5-10 mg. per cent should be maintained and the drug should be continued for several days after 3 consecutive daily smears have been negative, in order to forestall recurrences.

The great majority of writers recommend oral administration, and rightly so; for, as previously mentioned, in the presence of purulent secretion topical application of the drug would be ineffective. Nevertheless, there are American and foreign reports that local application is also effective. Using 0.5 per cent solution of sulfanilamide, Reins and Tibbetts[90] found that in 15 cases of gonorrheal conjunctivitis, smears became negative in an average of 6.8 days. However, the concomitant oral administration of the drug in some of their cases, because of the presence of the disease in other parts of the body, lessens the value of their claims for local efficacy. Panneton[91] achieved excellent results in several cases treated by topical application of sulfanilamide, but here also the value of the findings is questionable since smears were not taken at any time. Bruens[92] and Pillat[93] state that negative smears were obtained within 24 hours after repeated application of a 10 to 30 per cent sulfonamide preparation known as albucid. Thus far, therefore, no convincing proof has been offered for discarding the oral and adopting the local use of sulfonamides in this disease.

TABLE I.

EFFECT OF SULFONAMIDES IN GONORRHEAL OPHTHALMIA

Drugs	*No. of cases*	*Avg. No. Days Req. for neg. smears*
Sulfanilamide*	150	5.2
Sulfapyridine**	85	2.6
Sulfathiazole***	18	2.3

* References 64, 66, 67, 68, 81, 88
** 70, 72, 74, 75, 78, 79, 81
*** 81, 88

TRACHOMA

As a general rule virus diseases are not influenced by sulfonamides, yet there are several notable exceptions mentioned in the literature such as trachoma, inclusion blenorrhea, lymphogranuloma venereum and psittacosis. These 4 viruses differ from the typical viruses, according to Thygeson,[94] in that the inclusion bodies vary in size, are basophilic rather than acidophilic and stain readily with Giemsa preparation. Both experimental and clinical investigators disagree concerning the effects of sulfonamides in trachoma. Julianelle, Lane and Whitted[95] and Burnet, Cuenod, Nataf and Rousell[96] demonstrated the disappearance of inclusion bodies in sulfanilamide-treated cases. Richards, Forster and Thygeson[97] found that pooled epithelial scrapings of sulfanilamide-treated cases failed to infect baboons. In contrast, Julianelle and Smith[98] state that the original capacity to infect is not altered by sulfanilamide in vitro.

Regarding the value of sulfonamides in trachoma, the opinions of most clinicians fall into one of three groups:

(1) Those who believe that sulfonamides are curative or at least of great value. Heinemann,[99] Loe,[100] Gradle,[101] Thygeson,[102] Guyton and Woods[86] and others.

(2) Those who classify these drugs as useful adjuncts. Dik,[103] Lian,[104] Schwarz,[105] Szinegh,[106] Gifford,[63] Poleff,[107] Schmid and Saubermann,[108] and Sory.[109]

(3) Those who consider them ineffective. Wilson,[73] Busacca,[110] and MacCallan.[111]

While evidence is lacking that sulfonamides actually cure trachoma, most clinicians agree that there is improvement in subjective and to a lesser degree in objective symptoms. If improvement rather than cure be taken as the criterion of therapeutic effectiveness, the majority of writers are in essential agreement. Gifford[63] writes, "There seems to be no doubt that sulfanilamide is useful in trachoma, especially when corneal ulceration and acute inflammatory symptoms are present." There are many cases, however (29 out of 77 observed by Smith, Julianelle and Whitted, for example) which are resistant to sulfonamide therapy. How the improvement is effected is not clear. One group asserts that the beneficial results obtained in the treatment of trachoma are procured by elimination of the secondary infection. Spink[112] suggests that if this be true sulfapyridine would be superior to sulfanilamide, since the organisms most frequently present are pneumococcus, staphylococcus, etc. Others maintain that the drug does not act upon the superimposed infection, but upon the virus itself. Substantiation for the latter theory is offered by Thygeson,[102] who found that sulfanilamide brought about rapid improvement in trachoma, whereas there was little or no effect upon the accompanying organisms. It appears to the writer that since improvement occurs in related virus diseases (inclusion blenorrhea and lymphogranuloma venereum), which rarely are complicated by secondary infections, the evidence is in favor of those who believe that sulfanilamide acts directly upon the virus. Further, it is more than probable that the drug effects improvement by concurrent action upon both the virus and the secondary organisms. McKelvie, Kirk and Holder[113] depict the clinical sequence following the institution of sulfanilamide therapy: In the first stage the pressing symptoms such as pain, photophobia and lacrimation decrease. The second stage (one to four weeks) is characterized objectively by a gradual clearing of the cornea, and subjectively by an improvement of the visual acuity. Finally, the exuberant fleshy granulations decrease; follicular hypertrophy and old corneal scars, however, are improved little or not at all. The fact that old corneal scars remain uninfluenced by sulfanilamide emphasizes the urgency for early treatment.[97,114] One must not mistake the early and rapid improvement for a cure, because too early withdrawal of sulfanilamide may lead to recurrences. This danger is indicated in Cooper's[115] report of 34 cases in which 21, or 62 per cent, suffered recurrences in 6 to 30 months. His experience is not uncommon among clinicians.[116]

There are a few reports in the literature indicating that topical application [117,118,119,120] and subconjunctival injections[121] may be effective, but oral administration of sulfonamide in trachoma is the generally accepted method. In fact, Thygeson states that local therapy is entirely ineffective. Most effective results are obtained by intensive treatment for a short time rather than small fractional dosages over a protracted period.[102,86,122] Smith, Julianelle and Gamet,[116] in a critical review of the literature, found variations in dosages from 15 to 90 or more grains a day and in duration of treatment from a few days to more than 70 days. These factors may account for some of the differences in effectiveness reported.

Although marked improvement may be obtained by the use of sulfanilamide alone, the great majority of writers be-

lieve that results are far better when it is supplemented by local therapy in the form of copper or silver salts or grattage.

INCLUSION CONJUNCTIVITIS

Inclusion conjunctivitis, like trachoma, is a virus disease which responds favorably to sulfonamide therapy.[94,123,124,125,126] Thygeson found that the time for healing decreased from 3 or 5 months to 2 weeks in infants and only slightly longer in adults. Guyton obtained cures in 2 cases within 6 days. Sulfathiazole seems effective when applied locally, according to Thygeson. Epithelial scrapings become negative for inclusion bodies in 2 to 4 days (Thygeson, Allen) but in some instances inflammatory signs may abate before the disappearance of the inclusion bodies (Schmid and Saubermann). As in trachoma, papillary hypertrophy responds more promptly to therapy than follicular hypertrophy. Unlike trachoma, however, inclusion blenorrhea is rarely complicated by secondary infections, and we may assume with Thygeson that improvement must be the result of the action of sulfanilamide in interrupting the intracellular development of the virus.

LYMPHOGRANULOMA VENEREUM OPHTHALMITIS

Ocular infection with lymphogranuloma venereum is rare, but, whether it be conjunctivitis, keratitis or iritis a sufficient number of reports exist to indicate that it responds to sulfonamide therapy as rapidly in the eye as in other parts of the body.[127,128,129,130] Like trachoma and inclusion blenorrhea, the disease is caused by a virus. Stein[131] advises that ordinary cases be treated with the following dosages: 4 grams daily for 4 weeks, 5.3 grams daily for 3 weeks, or 6.6 grams daily for 2 weeks.

ACUTE CONJUNCTIVITIS

Although most of the organisms causing acute conjunctitivis are admittedly sensitive to these drugs, the necessity of using sulfonamides locally in acute conjunctivitis is questionable, if we may judge from reports in the literature.

In cases of acute conjunctivitis caused by Koch-Weeks organisms, Wilson[73] observed that the drugs did not shorten the period of morbidity. Thygeson states that sulfanilamide had no effect on H. influenzae, D. pneumococcus and staphylococcus complicating trachoma. Guyton in 18 cases of acute bilateral conjunctivitis, caused by Koch-Weeks bacillus, staphylococcus, streptococcus, and pneumococcus, noted that the infections in the eyes receiving a bland ointment cleared as rapidly as those treated with a sulfanilamide ointment. Schmid and Saubermann agree that the results of chemotherapy are unimpressive. In severe infections Guyton and Woods demonstrated that sulfonamides administered orally markedly decrease the healing time. However, the ordinary case of acute conjunctivitis is too mild and self-limited to require so drastic a measure as oral therapy.

BLEPHARITIS

The most frequent form of blepharitis is caused by staphylococcus. Treatment has been unsatisfactory until recently. The simple application of sulfathiazole results in a local concentration sufficiently high to combat this stubborn disease without danger of systemic toxicity. According to Thygeson[7] a dramatic response is obtained by using it 4 to 6 times daily in a 1 to 5 per cent ointment. Even better results were noted when it was used in conjunction with injections of staphylococcus toxoid. Allen[8] confirms this and states that in 40 patients treated with the combined method, 34 had negligible symptoms within 1 to 4 weeks. Since Zaytzeff-Jern, et al.,[6] report that sulfonamides with bacteriophage are highly effective against staphylococcic infection, is it not possible that a similar combination might be effective in this disease?

SERPENT ULCER

Since serpent ulcers are caused by pneumococcus, and rarely by other organisms such as Morax-Axenfeld, B. pyocyaneus, etc., all of which are sensitive to sulfonamides, one is justified in using these drugs. Reports are available showing the value of chemotherapy in this condition. Thus, Bailey and Saskin[132] procured remarkable improvement in 6 cases, Roggenkamper[133] obtained striking results in 15 cases, and deBascaran[134] reported comparable results in 10 cases of serpent ulcer treated with sulfonamides. However, in these reports, bacteriological examinations were either few in number or not mentioned. Other clinical observations have been made by Rosengren,[135] Chamberlain,[136] Guyton and others. It has been demonstrated experimentally and clinically that sulfonamides are effective in B. pyocyaneus ulcer. Joy[137] and Lepard[138] administered the drugs by the oral route, von Sallman[21] by iontophoresis and Robson and Scott[139] by drops. Joy concludes his excellent investigation by stating that "The oral administration of sulfapyridine sodium is effective in the prophylaxis and the treatment of experimental B. pyocyaneus ulcer of the rabbit's cornea and that the efficacy of the treatment varies directly with the promptness with which it is instituted."

In contrast to these good results, those obtained in experimentally produced pneumococcal ulcers were disappointing according to Chamberlain, who found that while sulfapyridine caused an improvement in the conjunctival condition associated with serpent ulcer the effect upon the cornea was unimpressive. In 2 cases of pneumococcal serpent ulcer observed by the writer, the lesions progressed in spite of early chemotherapy with sulfonamides, each finally requiring keratotomy. Explanation for the poor results observed in some cases of serpent ulcer may lie in the delay in applying the drug or in those factors retarding sulfanilamide action: the avascularity of the cornea, the use of local anesthetics, or the presence of secretion and detritus, as mentioned above.

CELLULITIS OF LIDS AND ORBIT

Sulfanilamide is effective in the treatment of streptococcic infections of the lids and orbit. Spink[112] states that to be effective in cellulitis, treatment should be instituted early and the blood concentration should be high. It must be remembered that pus should be drained when localization occurs so as not to inhibit the sulfanilamide action. The first cases of orbital cellulitis satisfactorily treated with sulfanilamide were reported by Goldenburg[140] and Szinegh,[141] the latter also obtaining good results in lid infections. Guyton observed 5 patients with lid and orbital infections; 4 showed beta hemolytic streptococci on culture. The infection began to subside within 24 hours after the institution of sulfanilamide therapy. A later report described an additional case terminating satisfactorily. Further substantiation of its effectiveness is offered by Surgist,[142] who reported 2 cases of orbital cellulitis in which rapid improvement followed drainage of purulent material and administration of sulfonamides.

SYMPATHETIC OPHTHALMIA

Despite its importance, there are relatively few reports of the effects of sulfonamides upon sympathetic ophthalmia. Gamble[143] presented before the Chicago Ophthalmological Society in May, 1939, a case which, while not cured, had been kept under control by sulfonamides. When an opportunity to examine the eyes histologically was afforded by the accidental death of the child, microscopic studies revealed that in spite of large dosages of sulfonamides and the satisfactory clinical appearance, the disease process had still been active. Colenbrander[144] reported an astonishing improvement in the sympathizing eye

in a proved case of sympathetic ophthalmia, treated with dagenan and a liver preparation. Gifford[145] has observed 6 cases of sympathetic ophthalmia, 4 of which responded unusually well.

ENDOPHTHALMITIS AND PANOPHTHALMITIS

As in other diseases accompanied by purulent matter and detritus, sulfonamides to be effective must be used early in endophthalmitis and panophthalmitis. However, even in ordinary clinical cases where the time of initial treatment frequently cannot be chosen, the results are not entirely discouraging. Thus, 3 out of 7 cases of endophthalmitis and panophthalmitis reported by Guyton terminated satisfactorily, and in 6 cases of endophthalmitis mentioned by Guyton and Woods, 3 healed rapidly, 1 healed with organization of the vitreous, and in 2 the drugs had no effect. The results in panophthalmitis reported by these authors are even more encouraging for in 9 out of 13 cases (or 69 per cent) complete healing occurred. (Table No. 2.) It is probable that the percentage of cures would have been even higher had all the staphylococcic infections been treated with sulfathiazole or sulfapyridine, rather than sulfanilamide.

SUMMARY AND CONCLUSION

1. Penicillin, tyrothricin and similar substances obtainable from molds, fungi and bacteria, while not yet employed clinically in ophthalmology, give promise of future usefulness.
2. Sulfanilamide and sulfapyridine given orally, or sulfanilamide used topically, or any of the 4 most commonly used sulfonamides employed with iontophoresis or combined with a wetting agent result in a chemotherapeutically adequate concentration in the ocular fluids and tissues.
3. The action of sulfonamides is known to be inhibited by secretion and detritus so commonly associated with ocular infection, by local anesthetics and by the factors inherent in the avascularity of the cornea.
4. Oral administration of sulfonamides frequently produces ocular indications of systemic toxicity.
5. Topical application of sulfonamides to the denuded cornea retards epithelial regeneration and promotes scarring.
6. Sulfonamides given orally are effective in ocular complications of erysipelas, gonorrheal ophthalmia, trachoma, lymphogranuloma venereum ophthalmitis, serpent ulcer, cellulitis of lids and orbit, endophthalmitis,

TABLE II

EFFECT OF SULFONAMIDE ON PANOPHTHALMITIS*

No. of Cases	*Organism*	*Drug*	*Effect*
2	H. Streptococcus	Sulfanilamide	Subsidence of infection in both eyes
3	Staphylococcus	Sulfanilamide	2 eyes destroyed 1 healed
2	"	Sulfapyridine	Complete healing
2	"	Sulfathiazole	Complete healing
1	Meningococcus	Sulfanilamide	Dramatic cure
1	B. pyocyaneus	Sulfanilamide	None
1	B. coli and Staphylococcus	Sulfamethylthiazole	Doubtful

* Data taken from Guyton and Woods

panophthalmitis and sympathetic ophthalmia.

7. Sulfonamides applied locally are effective in pyogenic dermatoses, infectious blepharitis, inclusion blennorrhea, and some forms of acute conjunctivitis.

8. In conclusion it may be stated that although the sulfonamides have proved to be the most important addition to the armamentarium of the ophthalmologist since the introduction of organic arsenicals, they should not be used indiscriminately nor be considered a panacea.

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CHEMOTHERAPY FROM THE STANDPOINT OF THE OTOLARYNGOLOGIST

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AFTER floundering around for several years we believe we have developed a definite rationale in the application of chemotherapy in otolaryngology.

One of the most important conclusions which has been reached is this, that it is no longer enough to be merely otolaryngologists in order to practice our specialty intelligently; we must be also chemotherapeutists. I do not mean expert bacteriologists or laboratory technicians; I mean we should know what laboratory work we need, when to obtain it and how to apply it.

The reason for this broader training is clear. The administration of chemotherapeutic agents in our field is very different from that in general medicine and pediatrics. Our dosage must be greater and continued over a much longer period. Hence the rules generally followed by the medical profession as a whole must be radically altered to fit our special needs.

At the same time it must be emphasized that chemotherapy does not supplant surgery: rather it must be considered an extremely valuable adjunct. To be used most effectively it should be administered during the early stages of the disease and in conjunction with immune sera, if available. The above statement does not deny the value of chemotherapy when used in later stages, even when severe complications have arisen, but in all cases we prefer to delay surgery until an optimum blood level has been established. The most gratifying results of this method of procedure are the practical elimination of emergency operations and a marked lowering of our mortality rate.

Realizing that any chemotherapeutic agent can reach an infected area only by way of the blood stream, it is obvious that enclosed pus, bone necrosis and infected thrombi (whether in the mastoid, frontal bone or calvarium) must be removed completely by surgery. This has been proved repeatedly by our results in treating osteomyelitis of the frontal bone and lateral sinus thrombosis. In these thrombophlebitic cases, and especially in cavernous sinus thrombosis, while heparin will not dissolve a clot already formed, it will prevent clot formation, thereby allowing the drug to penetrate the infected wall of the vein. This combination of the sulfonamides and heparin is responsible for the spectacular recovery of many cases of cavernous sinus thrombosis.

Of all the sulfonamides, we have found sulfadiazine the most effective and least toxic. It has the highest degree of penetration into the cerebrospinal fluid, making it very important in the treatment of meningitis of otitic or sinus origin. Sulfathiazole has the least degree of penetration and is the most dangerous of all the sulfonamides when given in the dosage and over the long period of time necessary in our specialty.

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In acute infections of the upper respiratory tract local treatment without systemic administration of the drugs has not been very convincing.

We have had limited experience with gramicidin, but so far it has seemed of no help in treating acute staphylococcic infections. For local use in open wounds we prefer sulfanilamide powder used sparingly. The same powdered drug has given excellent results in the treatment of chronic suppurative otitis media.

Following is a summary of the work done at the Massachusetts Eye and Ear Infirmary from April 1 to August 1, 1942, together with some private case reports and slides. During this service at the Eye and Ear Infirmary it was our routine to see that every patient whose case was surgical, with the exception of those undergoing tonsillectomies and minor nasal surgery, were given sulfadiazine to a blood concentration of approximately 10 mgm. per cent. In this group there were 17 radical mastoidectomies, 17 simple mastoidectomies, 27 radical antra and ethmoidal exenterations and 5 radical frontals.

We had a total of 123 surgical cases which included everything from cysts and tumors of the nasopharynx to foreign bodies in the oesophagus and lungs. The only fatality was a cavernous sinus thrombosis which we inherited from the previous service. At necropsy this case showed complete blocking of the cavernous sinus on both sides, superior longitudinal sinus, right sigmoid sinus and both petrosals on that side — demonstrating again my conviction that in order to cure cavernous sinus thrombosis treatment must be instituted before the various channels are completely blocked.

There were only 3 cases of toxic reaction out of 125 patients who had rather massive doses of sulfadiazine. Two suffered from ureteral colic, one of whom required catheterization and the other of whom was relieved by discontinuing the drug, forcing fluids by mouth, and taking fluids intravenously. The third patient suffered from a skin rash severe enough to necessitate discontinuing the administration of the drug. Nausea, which had been such a frequent and disagreeable accompaniment in the early use of the sulfonomides, has been conspicuous by its absence and rarely is the cause for elimination or reduction of the drug.

Some of the most gratifying results have been obtained in cases of acute sinus conditions. In my private practice out of 30 patients whom I treated for this condition, 28 of them were completely cured and two required minor surgical interference in order to promote frontal sinus drainage. This brings up another factor in the use of the sulfonamides which is of great value: that is, the safety with which surgery can be performed during the acute stages of the disease.

In conclusion, I wish to show slides which illustrate some of the more spectacular results following the treatment of acute sinusitis with the sulfonamides. They illustrate the cases before and after treatment.

Symposium on Chemotherapy

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CHEMOTHERAPY IN RELATION TO OPHTHALMOLOGY AND OTOLARYNGOLOGY WITH SPECIAL REFERENCE TO THE SULFONAMIDE COMPOUNDS

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BY INVITATION

SINCE the local disinfection of tissues by the topical application of chemical agents is just as much a part of chemotherapy as the specific treatment of syphilis with arsphenamine or of bacterial diseases with the sulfonamide compounds, chemotherapy in this sense has long played an important role in the treatment of many diseases of the eye, ear, nose and throat due to local infections. Until the discovery of the sulfonamide compounds, however, chemotherapy by local applications of the salts of silver and mercury, the aniline dyes, etc., was of limited value and especially in relation to otolaryngology. The discovery of the wide range of therapeutic effectiveness of the latter, however, in the treatment of various local and general diseases due to many of the pathogenic bacteria, including a few due to the viruses, constitutes one of the most remarkable chapters in the history of therapeutic science, outranking in importance even the discovery of salvarsan itself.

Time does not permit me to review the large literature which has accumulated on the use of the sulfonamide compounds in relation to ophthalmology and otolaryngology. But even if it were otherwise, I would hesitate to pass critical judgment upon it since I am neither an ophthalmologist nor an otolaryngologist. As a chemotherapeutist, however, I may be permitted to pass in brief review what I believe are some of the important principles involved, along with their practical applications, which I hope may be of interest and clinical value.

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CARDINAL PRINCIPLES

In the first place the indiscriminate use of the sulfonamide compounds is to be regretted. This is not based as much upon the possibility and probability of various pathogenic bacteria acquiring resistance to them by their frequent use in the treatment of minor infections in individual patients as upon the fact that it breaks one of the cardinal principles in therapeutics. Indeed, I know of no evidence conclusively proving that the frequent use of the sulfonamide compounds in the treatment of minor infections runs the risk of rendering them ineffective in the treatment of severe infections, should such subsequently develop, by reason of acquired resistance to their bacteriostatic and bactericidal effects, although it is fairly well established that different strains of the same micro-organism may possess a higher natural resistance to them than normal. Fortunately this is comparatively uncommon but always disturbing although switching over to the use of another compound usually solves the difficulty in practice.

In the second place, the sulfonamide compounds are not always a short-cut or royal road to treatment. This is particularly true in severe infections. In other words, clinical experience has shown that the physician and surgeon cannot afford to omit anything else of proven value in treatment, with special reference to prompt and adequate surgical drainage whenever possible. The same applies to the simultaneous use of blood transfusions and those immune sera of known therapeutic value. Indeed, since it appears that the effectiveness of the sulfonamide compounds is mainly due to their crippling effects upon the proliferative activities of bacteria with the final disposal of the latter by immunological mechanisms and especially phagocytosis by the cells of the reticuloendothelial system aided by antitoxins, opsonins and agglutinins, I am always in favor of using appropriate immune sera for furnishing the hard pushed patient with these antibodies and especially in the treatment of the septicemias and the acute suppurative meningitides. Furthermore, it is not unlikely that the simultaneous administration of well prepared autogenous vaccines based upon skilfull bacteriological examinations for the purpose of securing the organism or organisms of primary infection or, otherwise, of well prepared stock vaccines, may be helpful in the treatment of some of the bacterial diseases and especially those of the ears, nose and throat including the nasal accessory sinuses if it is true, as I believe it to be, that specific antibodies may increase the therapeutic effectiveness of the sulfonamide compounds.

In the third place, due emphasis should be placed upon the advisability of early treatment and the administration of adequate doses. Here again there is a close analogy between chemotherapy and serum therapy. For example, we have long since learned that the earlier adequate amounts of antitoxin are administered in diphtheria, the better the prognosis. The same is true in sulfonamide therapy not to mention the fact that the sooner appropriate treatment is instituted in early syphilis the greater the chances of complete recovery. It is true that the sulfonamide compounds are not without possible toxic effects due to their acetylation or conjugation, but after all the great majority of the latter are of a minor character along with the fact that in general terms the sulfonamide compounds are of remarkably low toxicity. In other words, delay in treatment and timidity in dosage are not infrequently the primary causes of failure of sulfonamide therapy and especially in the treatment of severe and dangerous infections.

Whenever possible oral administration is probably always to be preferred, with the administration of double the usual amount for the first one or several doses. Otherwise parenteral administration is possible with most of the sulfonamide compounds whenever necessary or advisable. Thus 0.8 per cent solutions of sulfanilamide may be given subcutaneously, intravenously or intrathecally, neoprontosil intramuscularly or the soluble sodium salts of sulfathiazole, sulfapyridine or sulfadiazine intravenously. Not infrequently a combination of oral and parenteral adminstration is advisable, as is likewise a combination of oral and local administration, to which further reference will be made.

Fortunately the dosage of all the sulfonamide compounds is the same. Under the conditions, relatively simple rules suffice for their administration subject to change in different diseases and individual cases. In the treatment of severe infections, with special reference to the septicemias and meningitides, I usually calculate the total dose for 24 hours on the basis of 1 grain per pound of body weight. This total is divided into 6 parts with a dose given every 4 hours, day and night, by oral or parenteral ad-

ministration or a combination of the two. In less severe infections the total amount per 24 hours is based upon ½ grain per pound of weight, divided into 6 parts with a dose given every 4 hours, or into 4 parts with a dose given every 6 hours. Dosage in the case of minor infections and especially those of a chronic character is usually based upon ¼ grain per pound of weight divided into 4 parts with a dose every 6 hours. In most instances I usually administer at least 5 to 10 grains of sodium bicarbonate with each dose in an effort to prevent nausea and vomiting due to acidosis. In febrile patients I believe that the compounds should be administered until the patient is fever-free for at least 72 hours before dosage is reduced.

Because of its harmless character, no attention need be paid to cyanosis, but since it is likely to occur when large doses are given, it is always advisable to warn the patient or the family of its probable occurrence. I believe, however, that all ambulatory patients should be warned of the possible occurrence of disorientation and dizziness and especially in relation to the hazards of driving automobiles. Needless to state, blood examinations at frequent intervals for possible hemolytic anemia and leukopenia are advisable when large doses are administreed. The former is not dangerous, but if the latter is ignored or overlooked it may progress to agranulocytosis, which constitutes one of the gravest of complications in relation to sulfonamide therapy.

Natural allergy to a sulfonamide compound occurs, but is fortunately rare. It is usually expressed by high fever with skin rashes following the first dose. Acquired allergy is much more frequent and, in my opinion, is usually responsible for drug fever sometimes occurring about 10 days or longer after continuous sulfonamide therapy. It should never be ignored, as it is sometimes the forerunner of the dreadful agranulocytosis. Fortunately, an individual allergic to one sulfonamide compound may not be allergic to another in case a continuation of treatment is necessary or advisable. A simple and safe test is the oral administration of 5 grains with hourly temperature readings over a period of at least 4 to 6 hours.

It is also advisable to examine the urine for blood when large doses are being administered. Hematuria is usually due to the irritation of the uriniferous tubules by crystals of the conjugated or acetylated compound. This is always most likely to occur in the case of sulfapyridine, sometimes upon the administration of sulfathiazole, but only rarely in the case of sulfanilamide, neoprontosil and sulfadiazine. The gross appearance of the urine may be insufficient for its detection, as hematuria may be present with its discovery only by microscopical examinations. In this connection it is to be remembered, however, that the sediment obtained by centrifuging 15 cc. of normal urine frequently contains from 2 to 4 erythrocytes per microscopic field so that, contrary to widespread impression, the presence of a few erythrocytes in individuals under sulfonamide therapy does not necessarily constitute hematuria.

Unfortunately not all human beings absorb the sulfonamide compounds equally well from the gastro-intestinal tract. Consequently, examinations at intervals for the determination of free compound in the blood is advisable, especially in severe infections, to insure adequate concentrations by alterations in dosage or the method of administration. These are not always easily made in private practice, but when necessary at least one estimation should be made after the first 24 hours of treatment, the blood being taken midway between any 2 doses. In ambulatory cases, however, blood concentration determinations may be safely omitted unless there are reasons for suspecting deficient absorption.

In view of the importance of blood concentrations of free compounds, I lay great emphasis upon the importance of making sure that the patient, or those responsible for him, understands the importance of taking a sulfonamide compound day and night at exactly the intervals prescribed by the physician.

BACTERIOLOGICAL DIAGNOSIS IN RELATION TO SULFONAMIDE THERAPY

As previously stated, the indiscriminate use of the sulfonamide compounds is inadvisable. Indeed, I believe that their use, and especially the choice of a compound for treatment, should be based upon bacteriological diagnosis more frequently than is commonly the case. No one can appreciate the possible mistakes in bacteriologic diagnoses more than I, and especially when it is remembered that no examination can be better than the laboratory conducting it. In this connection, however, it is only right and proper to point out that errors are not infrequently due primarily to the physician or surgeon making the smear or culture. Unless proper technic is employed for securing the organism or organisms responsible for infection, the purpose of the examination is defeated at the outset because a laboratory can only report what it finds instead of what the physician expects or desires. For example, such organisms as the streptococci, pneumococci and *B. influenzae* are not at all likely to grow on plain agar with the result that only staphylococci, diphtheroid bacilli, *B. pyocyaneus* and the like are apt to be found under these conditions. In passing, it is also worthy of emphasis that only positive blood cultures with all precautions against contamination are acceptable for establishing the diagnosis of septicemia. Indeed, a severe local infection without septicemia may present all the clinical manifestations of the latter on the one hand, while on the other, a positive blood culture may be a matter of surprise in minor primary tissue infections.

Unfortunately, normal bacterial floras can be very confusing in the interpretation of laboratory reports and especially in the case of examinations of the eyes, nose and throat. It is true that cultures of the normal conjunctivae are sterile in a small percentage of cases but usually show the presence of micro-organisms with special reference to staphylococci as well as, less frequently, streptococci, pneumococci, colon bacilli, *B. fecalis alcaligenes*, diphtheroid bacilli, *B. pyocyaneus*, *B. subtilis*, etc. Likewise cultures of the normal nose and throat will invariably show the presence of various types of staphylococci and streptococci as well as quite frequently pneumococci, *M. catarrhalis* and other Gram-negative diplococci, diphtheroid bacilli, *B. influenzae*, *B. friedländeri*, etc. Consequently, I maintain that no bacteriologic examination alone can be the basis for a decision on whether or not the tonsils are sufficiently infected for indicating or warranting their surgical removal. Nor need I pause to emphasize that smears showing many spirochetes and fusiform bacilli do not necessarily indicate that Vincent's angina alone is present since such may be only a secondary and relatively unimportant secondary infection in agranulocytosis on the one hand, or of a leukemia on the other. Indeed, I believe that the otolaryngologist should seldom accept a bacteriological diagnosis of Vincent's angina until a total leukocyte count at least has been made.

Under the circumstances the ophthalmologist and otolaryngologist should make smears and cultures as carefully as possible and, if necessary, under the guidance or advice of a bacteriologist. Final interpretation, however, is always the function and duty of the clinician as is true or should be of all laboratory examinations, including the serologic tests for syphilis. Acute infections are usually due to a single organism, where-

as chronic infections are usually due to 2 or more organisms. Once a decision is reached, I believe the choice of a sulfonamide compound for treatment may be as follows, named in the order of preference, when 2 or more are mentioned:

Staphylococcus infections: Sulfathiazole
Hemolytic streptococcus infections: Sulfanilamide, neoprontosil or sulfathiazole
Streptococcus viridans infections: Sulfapyridine
Pneumococcus infections: Sulfadiazine
Meningococcus infections: Sulfanilamide
Gonococcus infections: Sulfathiazole
B. friedländeri infections: Sulfadiazine or sulfapyridine
B. pyocyaneus infections: Sulfanilamide or sulfathiazole
B. coli infections: Sulfathiazole or sulfanilamide
B. influenzae infections: Sulfapyridine, sulfathiazole or sulfanilamide
B. tularense infections of the eye: Sulfathiazole or sulfapyridine
Trachoma: Sulfanilamide

It will be observed, therefore, that the sulfonamide compounds have a very wide range of therapeutic activity in the bacterial diseases. Unfortunately, they have proven of value in the treatment of but 2 viral diseases, namely, lymphopathia venereum and trachoma, if it is granted that the latter is due to a virus, as I believe most available evidence at the present time indicates may be the case.

Consequently, the sulfonamide compounds should be worthy of use in the treatment of the following diseases of the eye: infected wounds, ophthalmia neonatorum, hordeolum and marginal blepharitis, dacryocystitis, acute and chronic conjunctivitis and keratitis due to bacterial infections, scleritis and episcleritis, iritis and iridocyclitis, some cases of choroiditis, uveitis and panophthalmitis, orbital cellulitis and periostitis as well as trachoma in its early stages; likewise in the treatment of various bacterial infections of the ears, nose and throat as follows: erysipelas, furunculosis, acute and chronic otitis media, acute and chronic labyrinthitis and mastoiditis, lateral sinus phlebitis including its associated septicemia, acute and chronic sinusitis, acute tonsillitis and peritonsillar abscess, acute septic sore throat, acute and chronic pharyngitis and streptococcus, pneumococcus, *B. friedländeri* and *B. influenzae* meningitis. Indeed, I believe that they are likewise useful in connection with the treatment of the acute infectious "common cold," not because they are effective against infection with a virus which is now widely believed to be the primary cause, but against the secondary bacterial infections which inevitably follow due to lowered resistance and probably alone are responsible for infections of the accessory sinuses and the lower respiratory tract. Unfortunately the sulfonamide compounds are ineffective in the treatment of diphtheria except when a severe secondary infection with *Staphylococcus aureus*, hemolytic streptococcus or pneumococcus is present in which case they should be employed along with antitoxin. Furthermore, I commonly employ them in the treatment of scarlet fever with the hope of reducing the incidence of otitis media and mastoiditis, excessive cervical adenitis and glomerulonephritis; likewise in the treatment of pertussis and measles, not with the hope of influencing the primary infections due to their viruses, but with the expectation of preventing secondary bacterial infections of the ears, mastoid cells and lungs.

In this connection it should be stated, however, that it is now the consensus that the Koch-Week's bacillus of acute contagious conjunctivitis is the *Bacillus* or *Hemophilus influenzae*. As is well known, this organism is not the primary

cause of influenza but frequently an important organism of secondary infection in this disease with special reference to pneumonia, as well as being frequently responsible for local infections in the eyes, ears, nose and throat and of a severe primary type of meningitis. Under the circumstances it should be renamed for which I have proposed the designation *Bacillus* or *Hemophilus pfeifferi* after its discoverer.

I may also mention that chemotherapy has as yet nothing to offer of clinical value in the treatment of tuberculosis, although I do not doubt but that sooner or later an effective chemotherapeutic agent will be discovered. Nor has chemotherapy anything to offer in the treatment of infections of the eye due to *Leptospira icterohemorrhagiae*, the etiologic agent of infectious jaundice. Needless to state, however, chemotherapy with the organic arsenical compounds and bismuth has proven of great value in the treatment of syphilis and other spirochetal diseases of the eye, ear, nose and throat, to which further reference will be made shortly.

LOCAL TREATMENT BY THE SULFONAMIDE COMPOUNDS

Fortunately, the conjunctivae, as well as the mucous membranes of the ears, nose and throat, are highly tolerant to the local instillation of 0.8 per cent solutions of sulfanilamide and 5 per cent solutions of neoprontosil as well as to the introduction of finely divided powders of sulfanilamide, sulfathiazole or sulfadiazine. Without reasonable doubt this method of treatment has proven sufficiently encouraging to warrant continued use and further clinical trial. Personally, I am more hopeful of successful results following the introduction of very finely divided powders of any of these 3 compounds into the eyes, ears, nose and throat, including the nasal accessory sinuses, than I am of instillations of solutions of sulfanilamide or neoprontosil because of much more prolonged anti-bacterial activity. It is important, however, to use small amounts in order to avoid caking with consequent irritation and possible obstruction to drainage. Undoubtedly they slowly dissolve with the possibility of raising local concentrations of the compounds to a therapeutic threshold. Furthermore, the application of 5 to 25 per cent ointments of sulfathiazole in an oxycholesterol - petrolatum or other suitable base is likely to be more effective in the treatment of hordeola and furunculosis of the external auditory canal and nasal atrium than the local disinfectants hitherto employed. But I believe that such attempts at local disinfection by a sulfonamide compound should always be combined with its oral administration even though the total daily dose in chronic infections for prolonged administration may be no more than 1/7 of a grain per pound of body weight which, in the case of an adult weighing about 140 pounds, would mean 20 grains per day divided into a dose of 5 grains every 6 hours.

Apparently, therefore, the local application of any of these 3 sulfonamide compounds chosen on the basis of etiology, in conjunction with oral administration, is justified in the treatment of the infectious conjunctivitides as well as of trachoma. But, owing to the ease and frequency with which disinfectants may be introduced into the conjunctival sacs, the chemotherapy of conjunctivitis by local disinfection always has and probably will continue to be more successful than in the case of the ears and nose, with special reference to the nasal accessory sinuses, unless otolaryngologists succeed in working out methods whereby patients can apply local disinfectants sufficiently frequently and thoroughly as conditions demand. No wonder that local disinfection has so largely failed in the treatment of chronic sinusitis when it is remembered that 1 or 2

treatments per week mean that the contact between a disinfectant and the infected tissues is to be measured in minutes and probably even in seconds insofar as bactericidal concentrations of the former are concerned. Needless to state, solutions of the soluble sodium salts of sulfapyridine, sulfathiazole and sulfadiazine cannot be used because of their high alkalinity. But the very finely divided powders of sulfanilamide, sulfathiazole or sulfadiazine may be employed in small amounts without apparent irritation of the mucous membranes, the destruction of leukocytes (which are so important in phagocytosis) or injury of the cilia (insofar as the nasal and faucial mucous membranes are concerned). Nor do these powders have to be sterilized as upon dissolving, their solutions appear to be sufficient for the prevention of infection due to organisms introduced with them.

Personally, I think otolaryngologists may turn hopefully to the treatment of chronic sinusitis and otitis media by local insufflations of these powders introduced by the Lederman hard rubber insufflator or some similar instrument and especially if it is found possible for the patient to do this at least once a day with sufficient thoroughness. And since so many chronic purulent diseases of the rhino-oto-laryngological tract are apt to be due to mixed infections, I would like to suggest that a powder of such fineness as to pass a 40 to 100 mesh be made up of equal parts of sulfanilamide, sulfathiazole and sulfadiazine.

THE PROPHYLACTIC USE OF THE SULFONAMIDE COMPOUNDS

Furthermore, since the introduction of crystals of sulfanilamide or sulfacetamide into wounds and the peritoneal cavity has proven so successful in the prevention of infection, the ophthalmologist and otolaryngologist do well to consider carefully the possible prophylactic value of the sulfonamide compounds in many of the diseases due to infection of these parts.

Personally, I believe that the ophthalmologist should always attempt some disinfection of the eye before iridectomy or other intraocular operation even though the conjunctivae are normal with only the usual organisms like *Staphylococcus albus*, non-hemolytic streptococci or pneumococci present in cultures. At least it is well known that any of these organisms, as well as *B. coli*, *B. pyocyaneus* and even *B. subtilis* are capable of producing severe and even dangerous infections when introduced within the eye, even though they possess very low virulence, due to the fact that the humors and intraocular tissues possess very little natural resistance to infection. For this purpose he may choose any disinfectant which experience has taught him to use with confidence although the sulfonamide compounds quite naturally command attention in this respect. I also think that it is advisable to administer a sulfonamide compound orally for at least 5 or 6 days after every penetrating injury of the eye, in which case the dose for an adult may be 15 grains every 6 to 8 hours, day and night.

If clinical experience has taught otolaryngologists the types of cases of mastoiditis most likely to result in an extension of hemolytic streptococcus or pneumococcus infection to the meninges after operations, it seems to me only sensible and reasonable to try to protect the patient against these dreadful complications by starting the administration of a sulfonamide compound before and certainly immediately after operation. Indeed, the possible prevention of acute suppurative mastoiditis as a complication of otitis media by the prompt administration of a sulfonamide compound, chosen on the basis of the results of a bacteriological examination, also appears advisable, although it is true that it may mask a mastoiditis which,

however, should be detectable in most cases by a skilfull roentgenologic examination. In this connection it also seems to me that it may be advisable to insufflate a powdered sulfonamide compound on the tissues after intranasal operations and tonsillectomies not only because it may be helpful in the prevention of capillary oozing, but especially for the control of exacerbations of infection or the development of the inevitable secondary infections. Needless to state, I believe that this local disinfection should be combined with the oral administration of a sulfonamide compound for at least 3 to 5 days which, in the case of adults, may be 15 grains every 6 to 8 hours, day and night, with less for children according to body weight.

THE CHEMOTHERAPY OF SPIROCHETAL DISEASES

Up to this point I have confined myself to a discussion of the bacterial and viral diseases in relation to ophthalmology and otolaryngology but, in conclusion, no discussion could be complete without at least brief reference to the treatment of diseases due to spirochetes and especially syphilis.

It is true that some of the pathogenic yeasts and molds produce diseases of eyes, ears, nose and throat, but, unfortunately, chemotherapy has nothing of special value to offer in their treatment except the usual fungicides employed by local application in the treatment of the ringworms and thrush unless it be sulfanilamide in the treatment of actinomycosis in which disease it is reported as having yielded encouraging results in some cases. Fortunately, the pathogenic rickettsiae do not ordinarily produce diseases in relation to ophthalmology and otolaryngology, but even were it otherwise, chemotherapy has nothing to offer in their treatment. It is true, however, that chemotherapy has proven wonderfully effective not only in the treatment of malaria but other diseases due to animal parasites as well, although, fortunately, but few involve the eyes, ears, nose and throat and especially in the United States.

Insofar as syphilis is concerned, the treatment of the disease in the primary stage, characterized by chancres of the eyelids, nose or fauces, differs in no way from that of primary syphilis elsewhere in the body. Suffice to state that diagnosis should be made as promptly as possible, usually with the aid of dark-field examinations for *T. pallidum*, so that treatment may be instituted without delay. In this connection it is to be stated, however, that dark-field examinations are of little or no value in the diagnosis of chancres of the tonsils or oral cavity because it is impossible to differentiate between *T. pallidum* and *T. microdentium*, the latter commonly occurring in the saliva and especially in individuals with spiro-fusillar gingivitis. In this connection it is to be emphasized, however, that no ophthalmologist or otolaryngologist should undertake the treatment of early syphilis unless he is willing to use the continuous method over a period of about 15 months, employing alternate courses of an organic arsenical by intravenous, and bismuth by intramuscular injection, as recommended by the Clinical Cooperative Group.

As is true of clinically latent and chronic syphilis in general, however, ophthalmologists and otolaryngologists are properly urged to employ the serologic tests in both private and clinic practice for their detection more commonly than is generally the case. Needless to state, a negative history possesses but little value in excluding its possible presence. Furthermore, respectability is no bar to the penetrability of *T. pallidum*, so that the disease occurs among both the high and the low, irrespective of race, sex or social status. When properly conducted, the approved complement-fixation and flocculation

tests available at the present time possess a remarkable degree of both sensitivity and specificity. Unfortunately, however, falsely positive reactions occur in the best laboratories, although their incidence is largely in relation to the skill and experience of those who conduct them since no test can be better than the laboratory conducting it. Undoubtedly there is no one best serologic test for syphilis. Indeed, the results of all serologic surveys conducted by the U. S. Public Health Service in cooperation with the American Society of Clinical Pathologists have shown that the serologic diagnosis of the disease is best served by using routinely at least 2 or more tests possessing the maximum amount of sensitivity consistent with specificity.

Inevitably positive reactions are observed in individuals in whom the disease is not suspected clinically. Under the conditions they should not be ignored, but the tests should be repeated one or more times since repeated positive reactions may be, and usually are, the only evidence of the disease when its detection is not otherwise possible and at a time when the prompt institution of proper and adequate treatment may be of inestimable value to the individual. When different laboratories report varying results, it is not infrequently difficult to reach a decision but, in my opinion, this largely rests on the clinician rather than on the laboratory since the latter is doing all that can be reasonably expected by conducting the test or tests exactly as described by author serologists and reporting the reactions exactly as observed, rather than according to what the physician expects or desires.

Insofar as the treatment of chronic latent or tertiary syphilis occurring in ophthalmologic and otolaryngologic practice is concerned, it differs in no essential particulars from that of the disease elsewhere in the body. Unfortunately, however, it has not been found possible to standardize treatment as has been largely possible in the case of early syphilis. Indeed, every patient requires individual management and, unless the ophthalmologist or otolaryngologist has acquired special skill and knowledge in its treatment, it is better to refer the patient to some colleague who has both qualifications insofar as treatment is concerned. Certainly early syphilis is curable in the great majority of cases when appropriate treatment is given. The old dictum "once syphilitic always syphilitic" is no longer necessarily true. Even in chronic syphilis, the disease may be arrested and improvement amount to what has been called "clinical cure." It is important to treat the patient rather than his positive serologic reactions.

There are, however, 2 special types of chronic syphilis in relation to ophthalmology, namely, interstitial keratitis and primary optic atrophy, deserving of special mention in relation to treatment. As recently stated by Klauder and Vandoren,[2] best results are observed in the former by the administration of an organic arsenical compound along with bismuth in combination with fever therapy. Needless to state, the treatment of primary optic atrophy due to syphilis is discouraging at best insofar as restoration of vision is concerned although always advisable with the hope of preventing further progression of the optic neuritis as well as of retrobulbar neuritis present in some cases. It is now the consensus, however, that tryparsamide is inadvisable because the incidence of amblyopia following its administration in maximum doses is about 10 times greater than in those with normal eyes. Apparently intracisternal injections of arsphenamized serum constitutes the best routine therapy along with the oral administration of maximum doses of potassium iodide. Fever therapy conducted by inoculation with malaria, however, is always worthy of trial while the surgical relief of arachnoid ad-

hesions around the optic chiasm may be considered in some cases, as recommended by Hausman.[1]

In Vincent's angina local applications of neoarsphenamine in powder form or of 4 per cent solutions in 50 per cent glycerine are usually helpful, although in severe infections the compound should be given also intravenously or fuadin intramuscularly. The organic arsenical compounds have also proven highly effective in the treatment of yaws, pinta and the relapsing fevers, while being sometimes helpful in the treatment of those cases of rat-bite fever due to infection with *Spirillum minum*.

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1. Hausman, L.: Surgical Treatment of Syphilitic Optic Atrophy Due to Chiasmal Arachnoiditis, Am. J. Ophth. 24:119 (Feb.) 1941.
2. Klauder, J. V., and Vandoren, E.: Interstitial Keratitis-Standardization of Treatment, Ven. Dis. Inform. 22:307 (Sept.) 1941.

TRANSACTIONS

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High-Lights of the Forty-Seventh Annual Convention

The American Academy of Ophthalmology and Otolaryngology held its 47th annual convention at the Palmer House, Chicago, October 11-14, 1942.

Officers Elected — 1943

President.....James A. Babbitt, M.D.
Philadelphia, Pa.

President-Elect.....Lawrence T. Post, M.D.
St. Louis, Mo.

First Vice President..Sanford Gifford, M.D.
Chicago, Ill.

Second Vice President.Wesley Bowers, M.D.
New York, N. Y.

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Secretary for
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Secretary for Instruction—
Ophthalmology .....A. D. Ruedemann, M.D.
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Secretary for Instruction—
Otolaryngology .....Dean M. Lierle, M.D.
Iowa City, Iowa

Secretary for
Public Relations...Erling W. Hansen, M.D.
Minneapolis, Minn.

Chicago — 1943

Headquarters, Palmer House

Chicago has been selected as the city where the 1943 convention of the American Academy of Ophthalmology and Otolaryngology will be held.

Registration of Members and Guests

Registration in round figures, inclusive of members, guests exhibitors and ladies, totalled 1,750.

President's Reception

On Sunday afternoon, the president's reception was attended by more than one thousand members and guests. In the receiving line were Dr. and Mrs. Ralph I. Lloyd, Doctor and Mrs. E. C. Ellett, Dr. and Mrs. Frank Brawley, Mrs. Harry Gradle, Dr. F. J. Pinkerton and Dr. O. E. Van Alyea.

Honor Society

Drs. James A. Babbitt, Philadelphia, John J. Shea, Memphis, Tennessee, and Arthur J. Bedell, Albany, New York, were elected members of the Honor Society at its annual session.

Dr. Edward C. Ellett, of Memphis, Tennessee, was elected president of the society, whose members are elected in recognition of their scientific achievements and of their services to the Academy. Dr. Ellett will take office January 1, 1943. Dr. Thomas E. Carmody, Denver, was elected vice president.

Dr. Babbitt, emeritus professor of clinical otolaryngology at the University of Pennsylvania School of Medicine and associate professor of otolaryngology in the graduate school of medicine at the university, is president-elect of the Academy. Dr. Shea is a former vice president and member of the council of the Academy, a member of the American Board of Otolaryngology and former chairman of the section on laryngology, otology and rhinology of the American Medical Association. Dr. Bedell taught diseases of the eye at Albany Medical College for many years and is now emeritus professor. He has been honored for special research by the Medical Society of the State of New York and the section on ophthalmology of the American Medical Association.

Scientific Program

The joint session was held on Sunday afternoon, October 11, at two o'clock, at which time were heard addresses of welcome from Governor Dwight H. Green and Mayor Edward J. Kelly, and the President's Address.

The scientific program began with a symposium on *Chemotherapy*. Dr. John G. Bellows, Chicago, and Dr. Charles T. Porter, Boston, spoke on the subject from the standpoint of the ophthalmologist and otolaryngologist, respectively. By special invitation, Dr. John A. Kolmer, Philadelphia, spoke on the subject from the standpoint of the internist.

The scientific program was continued in the afternoon sessions, Monday, Tuesday and Wednesday, according to the schedule outlined in the program.

Meet Your Fighting Forces

The menu service dinner and "Meet Your Fighting Forces" program, held Tuesday evening, October 13, was an outstanding feature of the convention activities. Dinner was served in the Red Lacquer Room, and President Ralph I. Lloyd introduced the special guests, namely, officers of the Sixth Service Command of the U. S. Army, the Ninth Naval District of the U. S. Navy, the Ninth Naval District of the U. S. Coast Guard and the Ninth Naval District of the U. S. Marines, together with their wives. Doctor Kaspar Pischel, San Francisco, Guest of Honor, who was unable to be present, was made an honorary member of the Academy. Doctor E. C. Ellett, Memphis, Honor Guest, was the speaker of the evening. Awards of merit which consisted of plaqued certificates were presented to them.

The program "Meet Your Fighting Forces" was presented in the Grand Ball Room by the Army, Navy, Marine and Coast Guard units of the Chicago area. The Ball Room was filled to capacity, and the demonstration consisted of a swift and dramatic review of preparations for war by our fighting forces. The Navy Pier Band, under the direction of Chief Bandmaster Fred Norton, gave renditions of National anthems throughout the program. The Great Lakes Naval Training Station 50 Voice Choir under the direction of Lt. Cmdr. Hjalmer Hanson, also furnished music throughout the evening. The program consisted of an exhibition of flags by the Army, Navy, Marine and Coast Color Guard; explanation of a Garand rifle by a U. S. Marine; drill exhibition by

the U. S. Marines; hospital corps demonstration by the Great Lakes Naval Hospital; semaphore demonstration by the Naval Signal School, University of Chicago; Naval Aviation demonstration by the Cadet Selection Board, Chicago; Naval radio demonstration by Naval Training School, Chicago; breeches buoy rescue, by the U. S. Coast Guard; U. S. Army demonstration by the 7th Service Command of the U. S. Army; Aviation demonstration by the Air Force Sponsors of Chicago, Inc.; and induction of 100 recruits into the U. S. Navy by Lt. Comdr. David N. Goldenson.

The "Meet Your Fighting Forces" program was stimulated and largely carried out through the efforts of Dr. O. E. Van Alyea, of Chicago.

Dr. Lawrence T. Post President-Elect

Dr. Lawrence T. Post, professor and head of the department of clinical ophthalmology at Washington University School of Medicine, St. Louis, was named president-elect of the Academy at its annual business session Tuesday, October 13.

Vice presidents elected were Drs. Sanford Gifford, Chicago; Wesley Bowers, New York, and William Krieger, Poughkeepsie, New York. Dr. William L. Benedict Rochester, Minnesota, was re-elected executive secretary-treasurer.

Dr. James A. Babbitt, Philadelphia, emeritus professor of clinical otolaryngology at the University of Pennsylvania and associate professor of otolaryngology in the graduate school of medicine at the university, is the present president-elect. He will take office January 1, 1943, and Dr. Post will become president January 1, 1944.

Other new officers elected are Drs. Oliver E. Van Alyea, Chicago, secretary for otolaryngology, and Algernon B. Reese, New York, secretary for ophthalmology. Dr. Dean M. Lierle, Iowa City, was re-elected secretary for instruction in otolaryngology, Dr. Albert D. Ruedemann, Cleveland, secretary for instruction in ophthalmology, Dr. Erling W. Hansen, Minneapolis, secretary for public relations, Dr. Secord H. Large, Cleveland, comptroller, and Dr. Carl H. McCaskey, Indianapolis, member of the council.

Dr. Post, a native of St. Louis, graduated from Johns Hopkins University School of Medicine, Baltimore, in 1913. He entered private practice in St. Louis in 1915 and is at present ophthalmologist-in-chief to Barnes Hospital and St. Louis Children's Hospital in addition to his teaching activities. He was editor of the American Journal of Ophthalmology from 1924 to 1941 and editor-in-chief for the last ten years of that period. During World War I Dr. Post was surgical director of Mobile Hospital No. 4 in the American Expeditionary Force. He has served as president of the St. Louis Ophthalmological Society and chairman of the section on ophthalmology of the American Medical Association and the Southern Medical Association. In addition to the societies mentioned, Dr. Post is a member of the American Ophthalmological Society, Missouri State Medical Association and a fellow of the American College of Surgeons.

Scientific Exhibit Awards

The Academy presented five awards to participants in its scientific exhibit in connection with the annual session. Two awards went to exhibits on diseases of the ear, nose and throat and three to exhibits on diseases of the eye.

Dr. Theodore L. Terry, Boston, received the award for originality for his exhibit of drawings and illuminated transparencies showing abnormal development of the eyes in infants born

prematurely. The award for excellence of presentation was given to Dr. Abraham L. Kornzweig, of Mount Sinai Hospital, New York, for an exhibit showing how certain parts of the eye (cornea, sclera and the junction of these two parts) develop in an unborn child. Dr. Donald J. Lyle, Cincinnati, was honored with an award for the exhibit "of greatest value to medicine." Dr. Lyle's exhibit dealt with the relationship of the eye to the brain, showing the effect of certain diseases of the brain on the eyes. The diseases studied included tumors, diseases of the blood vessels and various types of inflammations. Dr. Lyle showed how the eye is a "show window" revealing conditions affecting the brain and how study of the eye is of great importance in diagnosis of diseases of the brain.

In the field of diseases of the ear, nose and throat, Dr. Anderson C. Hilding, Duluth, Minnesota, received the award for originality of research. His exhibit, which accompanied a paper presented in the scientific sessions, showed that deaths from severe asthma and other respiratory diseases may be due to failure of the small hair-like processes which normally cover the mucous membrane in the upper part of the respiratory system. Dr. Hilding has found that in persons with asthma, pneumonia and other respiratory diseases, the normal membrane has been replaced by one with few of the cilia or hair-like processes. When this happens the mechanism for removing secretions is insufficient, with the result that the bronchial tubes become filled with secretion and death results from asphyxiation. The exhibit in this section considered to be of great value to medicine was a study of the appearance of normal and diseased tissues in the ear, nose and throat, presented by Drs. Maurice F. Snitman and Irwin G. Spiesman, of the University of Illinois College of Medicine, Chicago.

PROGRAM COMMITTEE MEETING

The Program Committee will meet in Chicago in January, 1943. Requests for participation in the scientific program and other branches of the Academy's program should be in the hands of the respective secretaries before January.

O. E. Van Alyea, M.D., Chicago, Secretary for Otolaryngology — scientific papers, motion pictures and scientific exhibits.

Algernon B. Reese, M.D., New York, Secretary for Ophthalmology — scientific papers and motion pictures.

In Memoriam

Harry B. Davis.....Kansas City, Missouri
November 23, 1941

Louis S. Dunn.....Philadelphia, Pa.
June 16, 1942

Dred Rumley Dorente.....Ada, Oklahoma
June 17, 1942

Nelson Shelly Weinberger.....Sayre, Pennsylvania
June 28, 1942

Stanley G. Miner.....Detroit, Michigan
June 28, 1942

Richmond McKinney.....Memphis, Tennessee
June 30, 1942

Henry William George.....Middletown, Pennsylvania
July 7, 1942

George S. Row.....Indianapolis, Indiana
July 8, 1942

William Fowler .....Detroit, Michigan
July 13, 1942

Robert Emmett Buckley.....New York, New York
July 25, 1942

Alice Gertrude Bryant.....Boston, Massachusetts
July 25, 1942

Carl Charles McClelland.....Detroit, Michigan
July 29, 1942

Edward Powers Norcross.....Highland Park, Illinois
August 5, 1942

Frank Vinsonhaler.....Little Rock, Arkansas
September 1, 1942

Donald M. Campbell .....Detroit, Michigan
August 25, 1942

C. C. Eves.....Philadelphia, Pennsylvania
September 22, 1942

Joseph C. Beck.....Chicago, Illinois
October 21, 1942

Allen Greenwood.....Boston, Massachusetts
October 24, 1942

Society Proceedings

The Association for Research in Ophthalmology, Inc.
Thirteenth Scientific Meeting

June 9, 1942
Atlantic City

Algernon B. Reese, M.D., New York, N. Y.
Chairman

C. S. O'Brien, M.D., Iowa City, Iowa
Secretary-Treasurer

A QUANTITATIVE STUDY OF THE CELLS AND FIBERS IN THE NUCLEUS-NERVE COMPLEXES OF THE FOURTH AND SIXTH CRANIAL NERVES

Robison D. Harley, M.D., Ph.D.
Mayo Clinic, Rochester, Minn.

(Paper read by W. L. Benedict, M.D.)

There is clinical and physiological evidence that there must be connections between the different oculomotor nuclei because of the smooth interplay and reciprocal action that exists between the eye muscles, not to mention further nervous pathways connecting the nuclei of the midbrain to the hindbrain and even the spinal cord. Our problem is the location of the cell stations of these oculomoter connections. In this study the numerical relationship between fibers and cells of the fourth and sixth cranial nerves and nuclei has been established. If there are fewer cells than motor fibers a part of the cells of origin must lie outside of the circumscribed masses of gray matter which are the respective nuclei. More cells than fibers, on the other hand, would indicate that there must be nervous connections other than to the peripheral nerve, the fibers of which are supplied at least by some of the cells of these nuclei.

The nerve and nucleus specimens were collected from 32 normal human brains but due to multiple samples 113 fourth nerve sections, 77 sixth nerve sections, 53 fourth nuclei and 49 sixth nuclei were actually analyzed. Each nerve was studied to determine the number of myelinated nerve fibers and also the total number of fibers. The nuclei were sectioned serially at 10 microns and every tenth section was analyzed for the total number of motor cells present.

It was found that the mean number of cells for the nucleus of the fourth nerve was 2383 and the mean number of fibers was 2147. For the sixth nerve the mean number of cells of the nucleus was found to be 6154 and the mean number of fibers 5176. No significant difference was found between the right and left sides as respects both the number of cells and the number of fibers. The number of cells in the nuclei was found to exceed the number of fibers in the corresponding nerve in almost all instances. The cell-fiber ratios were also studied and the mean values for the same four nucleus-nerve complexes were 1.11, 1.15, 1.21, and 1.19. The relationship of cells to fibers was also studied graphically with the aid of fitted regression lines.

The number of unmyelinated fibers, as determined by the difference between the total number of fibers and the number of myelinated fibers, was 150 for the fourth and 440 for the sixth nerve. The average length of the nucleus for the fourth nerve was 2.4 mm. and for the sixth nerve 3.3 mm. The mean cross-sectional areas for the corresponding nerves, respectively, were 0.22 and 0.59 sq.mm.

The fourth and sixth cranial nerves belong to the type of mixed nerve whose afferent fibers convey pain and the proprioceptive sense.

Based on the work of Bjorkman and Wohlfart and Zotterman we may estimate that at least 39 per cent of the fibers of the fourth nerve and 22 per cent of the sixth nerve fibers are sensory. Including these figures into our work we find about 85 per cent more cells than motor fibers for the fourth nucleus- nerve complex and, similarly, over 50 per cent more cells than motor fibers for the sixth complex.

Since there are clearly more cells in the so-called nuclei than motor fibers in the peripheral nerves it is logical to assume that the motor nuclei of the fourth and sixth cranial nerves send fibers not only peripherally into these nerves but centrally to other regions, such as other cranial nuclei, probably via the median longitudinal fasiculus. Some of the association mechanism (association centers) may thus be within the confines of the so-called nuclei of the ocular nerves.

DISCUSSION

H. J. SMITH, M.D., Chicago, Ill.: I would like to ask Dr. Benedict if there was any proof found for the fact that some of these nerve cells in the fourth nerve, where they could not find fibers connected with them, may not have been for correlation with the nerve of the optical side. I have not been able to find any such evidence in the literature, but it occurs in so many parts of the nucleus of the third nerve that I wonder if there could be any decussation with the nucleus of the opposite side in the case of the fourth. I don't know anything about the sixth in that regard, but I suspect that there are none.

W. L. BENEDICT, M.D., Rochester, Minn.: I think that Dr. Harley did not assume terminal association. His work had to do with the actual count. On the other hand, the deviation, the individual variability between the right and the left side, was statistically insignificant, and there appeared to be nearly equal excess of cells over fibers on both the right and the left side, so it is quite possible that there was a direct relationship, although that is purely an assumption. How many of the fibers might go to the nucleus of the third nerve or the sixth nerve nucleus, or whether they would go to some other nucleus, really, we haven't any way of knowing just what that relationship is, but it is presumed that certainly there is a sufficient excess of cells over fibers for that to be possible.

In going over some of the specimens Dr. Harley rather felt that probably some of those nucluei were idle and that they had no connection with any other source. At any rate, he was not able to trace, except in myelinated fibers, any other group of fibers to these cells. They simply exist there, but what connection they have, he was not able to determine.

FREDERICK H. VERHOEFF, M.D., Boston, Mass.: In other words, we can't draw any conclusion from it?

DR. BENEDICT: No.

DR. VERHOEFF: You spoke of proprioceptor fibers. The evidence is that there is no poprioceptor for the ocular muscles. The work that was done by Ludvigh and Irvine indicated very clearly that the old idea that the knowledge of position of the eye was not dependent on a proprioceptor was true, and, therefore, you don't have to consider proprioceptive fibers in this connection at all.

DR. BENEDICT: It is assumed that those fibers, that were not myelinated motor fibers, were sensory, but he did not say whether they were proprioceptive or not.

THE INTRASCLERAL VASCULAR PLEXUS AND ITS RELATIONS TO THE AQUEOUS OUTFLOW

Manuel Uribe Troncoso, M.D.
New York, N. Y.

The anatomical arrangement of the vessels in the angle of the anterior chamber has a considerable influence on the movement and the rate of the aqueous outflow.

In previous articles I have shown that there is a great difference in the anatomic building of this region between lower mammalia and primates. In rodents and ungulata there is a cleft between the sclera and the outer side of the ciliary body which is filled with aqueous and communicates with the anterior chamber through the socalled Fontana spaces. To this cleft I have given the name of "cilioscleral sinus." In the rabbits it is made of large spaces, but in ungulata it is filled up with a peculiar spongy tissue. On both sides of the cleft there are numerous vessels. On the outer wall there is a rich vascular network in the thickness of the sclera called "intrascleral plexus." Some of its branches are very superficial, their endothelial wall coming in direct contact with the aqueous, through the network of the trabeculum. These veins have been compared to the Schlemm canal in man, but, as they have different anatomical characteristics, should properly be called "trabecular veins." In these animals the whole cilioscleral sinus performs the physiological function of the Schlemm canal in primates. On the ciliary body side of the ceft there are also vessels which come in contact with the aqueous.

In carnivora the sinus is filled up entirely with numerous fine strands which are tendons of the ciliary muscle. They are closely packed and to facilitate aqueous absorption, the intrascleral vascular plexus is exceptionally rich. It is lodged in large cavities inside the sclera: the Hovius ring.

In primates the anatomical arrangement is different. The ciliioscleral sinus has disappeared, due to the enormous development of the ciliary muscle. A new organ appears: the Schlemm canal, which is lodged in a special groove in the sclera above the spur. The whole intrascleral plexus has moved forward. It becomes connected with the Schlemm canal by a few branches called "collectors" (from 20 to 30 around the limbus) while the rest of the plexus has numerous richly interlacing branches. The Schlemm canal is really a small plexus in itself. Leber considered it a venous sinus; Swindle, one of the many veins of the intrascleral plexus. These opinions are not tenable, however, since by gonioscopy the Schlemm canal appears filled with aqueous and only rarely contains blood. Friedenwald has found fine arteries connecting directly with the lumen of the canal.

From the physiological standpoint the absorption of aqueous is different in lower mammals and primates. In the former the endothelial wall of the trabecular veins forms the blood aqueous barrier.

Absorption of aqueous takes place especially at the venous exits of the capillaries by a combination of hydrostatic and osmotic pressures.

In primates conditions are different; there is no blood-aqueous barrier. The Schlemm canal is normally filled with aqueous absorbed by a combination of hydrostatic and osmotic pressures. In the rest of the intrascleral plexus blood pressure is lower and aqueous passes directly into the blood through the collector channels. A slight amount of plasma leaks backward, normally into the canal; but if blood pressure is increased, red corpuscles may penetrate into it.

DISCUSSION

K. W. ASCHER, M.D., Cincinnati, Ohio: I think that the detection that the aqueous venous channels change continually with elimination of fluid out of the human eye is unexpected confirmation of the whole concept of the latest theory and especially confirmation of the most interesting anatomical and physiologic details described by Dr. Troncoso. We will discuss more details this afternoon, bringing some slides which show the physiologic and anatomic behavior of this fluid elimination.

CORNEAL PERMEABILITY
Factors Affecting Penetration of Drugs Into the Cornea

Kenneth C. Swan, M.D., and
Norman G. White, M.S., Iowa City, Iowa

Passage of drugs through the cornea into the globe is generally considered to be a process of diffusion. The rate of diffusion is believed to be affected by variations in osmotic pressure, pH and concentration of the test solution as well as by the condition of the surface epithelium. Disturbances of the surface epithelium are known to increase corneal permeabilty to drugs. In addition, it has been assumed by some authors that relative permeability of the cornea to various compounds is in inverse relation to the size and complexity of the individual molecules. However, other physical properties of compounds have received little consideration in corneal permeability studies although lipoid solubility, surface activity, electrical charges and diffusion coefficients are known to affect the penetration of substances into the various plant and animal cells. The recent use by O'Brien and Swan of a surface tension reducing vehicle, zephiran, to increase absorption of carbamylcholine (doryl) suggests that at least one of these factors is of importance in relation to the cornea. These authors suggested also that the relative impermeability of the cornea to choline derivatives might be explained by the difference in physical properties of these compounds as compared to those of the alkaloids.

To study and correlate these possible new factors with older concepts required investigation of the entire problem of corneal permeability. Factors affecting the rate of penetration into the cornea were studied as an initial phase. Other phases of corneal permeability will be considered in subsequent reports.

The relative rates of penetration into the cornea of a group of naphthalene derivatives of similar molecular structure were determined and correlated with the physical properties of the compounds and their solutions. These compounds were selected for study because it was possible to change their physical properties by varying the number and position of polar amine and sulfonic acid groups on the non-polar naphthalene nucleus. Moreover, they were fluorescent, permitting biomicroscopic study of the penetration into the ocular tissues as well as quantitative

chemical analyses. In addition, relative rates of penetration into the cornea of a group of local anesthetics were investigated. The anesthetics selected were similar to each other in molecular weight and structure but had varied physical properties in accordance with changes in relationship between the non-polar and polar groups. Moreover, the physical properties of the free anesthetic bases differed considerably from those of the salts, permitting another type of correlation to be made between physical properties and rates of penetration.

The various test substances varied in rate of penetration into the cornea. Several of the relatively non-polar compounds penetrated many times more rapidly than relatively polar compounds of the same chemical group. Superficial lesions of surface epithelium decreased the differences in penetration rates indicating that the epithelium acted as a membrane with differential permeability. This differential permeability appeared to be closely related to the physical properties of the test substances. Physical properties of the drug vehicle were found to be of importance also. Possible therapeutic applications of these studies are discussed.

DISCUSSION

JONAS FRIEDENWALD, M. D., Baltimore, Md.: I believe that Dr. Swan's work has brought the problem of the permeability of the cornea in line with the permeability of many other organs. I think that his statement of the whole problem has greatly clarified the matter.

It seems to me, however, that the emphasis on the polar as against the non-polar structure, as if these were two opposite and antagonistic actions, is not quite clear, and I believe that Dr. Swan emphasized that beyond what he actually intended to in his paper.

In order that a substance should go through a lipoidal membrane, which is surrounded on both sides by an aqueous face, it has to have affinities both to the water and to the lipoidal portion, so that the maximum penetration is obtained by substances which have a balanced degree of polar and non-polar characteristics. Substances that are wholly polar, like inorganic salts or glucose or urea, have, as Dr. Cogan and his co-workers have shown, very little power of penetration through the corneal epithelium. On the other hand, substances that are wholly lacking in polar groups also have very little penetration. Only those substances which combine these two effects are able to get through both faces of this tissue.

That is a characteristic which has been found in a great many other organs, and perhaps the most classical study on these problems has been done by Amberson and Hoeber, who studied the penetration of various substances through the salivary glands, and who found that starting from a substance that is wholly non-polar, its permeability, its penetrating power, could be increased by adding polar groups; and, starting from a substance that is wholly polar, its penetrating character could be enhanced by adding non-polar groups. Finally, if substances of a similar balanced structure are compared, then molecular size plays an important role.

I would like to ask Dr. Swan whether that interpretation of his data is correct.

DR. SWAN: That is correct. I am sorry if I gave the impression that polar and non-polar are two separate entities. They are not. In this group of naphthalene derivatives that we studied, the middle group of compounds had both characteristics, and I appreciate what Dr. Friedenwald added to the statement in clarifying that.

SANFORD R. GIFFORD, M.D., Chicago, Ill.: What conclusions can be drawn as to the penetration of alkaloids not only into the cornea but into the aqueous in various media?

DR. SWAN: That, I think, we will have answered in the second paper. I mentioned the preliminary experiments. We found that the distribution of compounds in the aqueous was not a direct index of penetration across the epithelium. For instance, in the living eye, the blood supply to the iris, the activity of the iris, the distribution of the drug in the lens and in the vitreous, as well as to the anterior sclera, complicate the matters. For that reason, we separate the two problems, and the second phase of this work is the factors determining the concentration of drugs in the aqueous as compared to the cornea and other tissue.

CLYDE A. CLAPP, M.D., Baltimore, Maryland: It has been recently stated that drops instilled on the upper surface

of the cornea are absorbed more readily than when placed on the lower conjunctival tract. Does your work clarify that statement at all?

DR. SWAN: I think that point was clarified by Laquer in about 1876. He demonstrated that absorption from the conjunctival culdesac is primarily through the cornea as contrasted to the conjunctiva. Later on, Antonifon repeated that work, blocking off the conjunctiva (I have forgotten the name of the material, but I think it was sodium chloride) so that only the cornea was exposed. As I recall, he found that the ratio was nine to one in favor of the cornea; that is, 90 per cent of the material was transported by the cornea; so that instilling a drug over the cornea at the top of the cornea would bring more of the drug into contact with the corneal surface.

FIBROBLASTIC OVERGROWTH OF PERSISTENT TUNICA VASCULOSA LENTIS IN INFANTS BORN PREMATURELY

III. Studies in Development and Involution of the Hyaloid Artery and Tunica Vasculosa Lentis

T. L. Terry, M.D., Boston, Mass.

Study of fibroblastic overgrowth of tunica vasculosa lentis in infants born prematurely has led to interest in the development and regression of the hyaloid artery and tunica vasculosa lentis system. This vascular system had been studied in serial sections of eyes from human foetuses of various ages. India ink and other injections of the system have been made of foetal and postnatal rats, cats, pigs, and pounch young opossums. A few non-viable human foetuses have also been injected. A study has been made of the eyes of premature infants to determine the normal status of tunica vasculosa lentis following premature birth.

From this variegated material many interesting observations have been made. In disagreement with Mann's findings, the vessels of the pupillo-capsullary portion of tunica vasculosa lentis at early stages have no tendency to avoid the center of the pupillary area. Suggestions of parallelism between the main branches of the hyaloid artery and the Y suture of the lens are found in some cases. Simplification and reduction in number of the vascular pattern in the region of the equator correspond to the valleys of the ciliary processes which must indicate that at one stage the ciliary processes become more simple or the vessels have to adapt themselves to relatively few channels to reach the anterior surface of the lens. Instances of double hyaloid have been observed. Although there appears to be no reason to doubt that the hyaloid artery always carries blood toward the lens, it is probable that the pupillary-capsullo portion of the hyaloid system has both arterial and venous supply in common with the mesodermal portion of the developing iris, and therefore should be able to maintain patency even after the hyaloid artery is closed.

Factors causing first the development and later the regression of this vascular tree were discussed hypothetically. When the vascular system develops, the lens appears disproportionately large and is growing more rapidly than the rest of the eye. As the eye grows the hyaloid artery must grow or become stretched. As the need for a tremendous blood supply decreases, this stretching finally ends in breaking of the connection usually, but not invariably, near the nervehead. Instances of remains of the hyaloid artery reaching to the back of the lens testify to such variations. Disappearance of the annular vessel and later growth of the iris make the venous drainage of the vascular tunic more tortuous, and may tend to be somewhat of a stretching factor as well as offer a lessening of pressure difference through increased friction incident to the lengthening of the vessels. Based on Thoma's law vessels are kept open and grow because of the difference in blood pressure, disappearing when these differences have become considerably less. At early stages no aqueous is available to give nourishment to the lens. Development of aqueous gives another source of nutrition so that blood vessels are less necessary. Very striking shallowness of the anterior chamber, distortion of the ciliary processes, and failure to develop hydrophthalmos when glaucoma does occur in patients with the fibroplasia point to an abnormally meager production of aqueous.

GONIOSCOPIC STUDIES ON THE CANAL OF SCHLEMM

Peter C. Kronfeld, M.D.
H. Isabelle McGarry, M.D.
and Homer E. Smith, M.D.
Chicago, Illinois

The gonioscopic phenomenon, originally described by Salzmann, of a blood-filled canal of Schlemm is observed not infrequently as an accidental finding during routine examinations of non-glaucomatous eyes. The phenomenon is inconstant, difficult to reproduce and in many instances probably related to excessive squeezing on the part of the patient during the insertion of the contact lens or to pressure of the latter upon prominent anterior ciliary veins.

The present study deals with the constant and regular phenomenon of the appearance of blood in the canal of Schlemm in non-glaucomatous eyes following minimal to complete aspirations of aqueous.

After complete emptying of the anterior chamber the phenomenon is visible as soon as sufficient fluid has been reformed to permit gonioscopic examination, and persists until the tension has risen to a tonometric reading of about 15 mm. (Schiotz). During this period of hypotony the canal presents itself as a scarlet to purple-red band situated in the depth of the posterior two-thirds of the trabecular area. The band measures from one to two-thirds of the trabecular area in width. It is at first fairly uniform in density but breaks up into segments or islands toward the end of the hypotonic phase. Gonioscopically no leakage of blood from the canal of Schlemm into the anterior chamber could be observed.

In glaucomatous eyes this gonioscopic phenomenon presents strikingly different features, the study of which opens up a new avenue of approach for the investigation of the mechanism in the various forms of glaucoma.

DISCUSSION

MANUEL U. TRONCOSO, M.D., New York City: I think that gonioscopy is now not only an interesting method of practical examination of the eye, but also has been the way of promoting the consideration of and the research in the structure of the angle.

Dr. Kronfeld's paper can be studied in two ways: from the physiological standpoint and from the pathological standpoint. I am very glad that his considerations confirmed what I said this morning about the normal contents of the Schlemm canal, and that under pathological conditions of pressure, the canal fills up with blood. But he goes a step farther, and he has been able to trace the small pores, the small apertures, in the trabeculum from the Schlemm canal. That is very interesting because it brings those back to the old conception that the inner wall of the Schlemm canal is not a continuous membrane, but has some stroma or apertures that may bring aqueous inside of the canal. If that is due to preformed channels or simply due to the rupture of the endothelial wall of the Schlemm canal by the pressure increase inside by the blood, I can't tell, but that is something that the method of gonioscopy may be able to give us.

From the second point of view, from the pathological standpoint, Dr. Kronfeld's paper is very interesting, because it gives us the idea, the methods, the way of finding out if the Schlemm canal is closed in glaucoma or not. We know that in primary congestion, acute and subacute, the angle is almost always closed by synechia, partial or double. In this kind of glaucoma, there would not be any opportunity to find out by the method of Dr. Kronfeld because the canal is covered by the synechia, at least partially. But, on the other hand, in those glaucomas that he calls wide angle glaucoma, that we know as chronic glaucoma, sometimes we are in doubt whether it is glaucoma or not. In that method, by puncturing the anterior chamber and finding out if the canal fills up with blood (I do not understand how he sees it), he may find it partially closed or entirely closed. From that standpoint it is very interesting, and the method is not difficult. It does not empty the anterior chamber entirely. It takes only a small amount of fluid, and then gonioscopy is done again to find out if there is no blood in the canal, that the canal is closed. If there is some blood in the canal, if it is partially filled, then the canal is more or less partially or entirely open.

JONAS FRIEDENWALD, M.D., Baltimore, Md.: I should like to report in this connection on some experiments which have been performed by Dr. Sholz and myself, in which we tried to throw some light on the presence or absence of blood in the Schlemm canal of animals under normal conditions.

We used cats, dogs, rabbits and guinea pigs, and in order to avoid the possible loss of blood from the canal during the process of enucleation and fixation, we froze the eyes of the animals with liquid air without any previous local manipula-

tion. The front segment of the eye was then sawed off in frozen state and fixed by placing it in alcohol at temperatures far below zero. The sections then revealed the vessels which formed the base of the experiments.

In a number of animals we did anterior chamber punctures before the freezing manipulation, and under those circumstances, regularly found these vessels filled with blood.

These experiments, I think, have a bearing on and completely support Dr. Kronfeld's findings, and they also suggest that the anatomical differences which Dr. Troncoso described this morning between the lower animals and the primates have less functional significance than might be attributed to the variation in anatomical structure.

I should like to ask Dr. Kronfeld, also, whether he has done repeated punctures on some of these cases which show the bleeding phenomena, after a sufficient interval, that any mechanically ruptured pores might have had an opportunity to heal up. I think that such experiments might throw some light on whether these pores are normally present anatomical structures or whether they are merely the result of rupture from the mechanical manipulation.

K. W. ASCHER, M.D., Cincinnati, Ohio: There are so many valuable facts in the paper of Dr. Kronfeld that every chapter would warrant an extensive discussion. I would like to mention only a few points, and I ask Dr. Kronfeld to allow me to project once more the slide showing Salzmann's picture of the canal of Schlemm. Then I would like to show my slide, No. 6, which shows the aqueous veins.

(Slide) I would like to draw your attention to the fact that in the canal of Schlemm, the distribution of blood is apparently similar to the distribution of blood in the aqueous veins. Whenever you look at the picture of Salzmann showing the canal of Schlemm, you always want to doubt whether this (indicating) border of the blood band is really coincident with the border of the canal itself. I suppose that the canal of Schlemm reaches up to here (indicating) and that this whole feature might be the canal filled to one-half with blood and to one-half with aqueous humor, according to the findings in the aqueous veins.

I am very glad to find confirmation of this opinion in some words of Dr. Kronfeld, who said that in certain cases the ratio of blood aqueous is changing. It may be one-third, if I am right, or one-half or even less than one-third in glaucomatous eyes. That would mean that the blood level in the canal of Schlemm may change and that what we see is this (indicating). This is the canal of Schlemm.

Now let me see No. 6 of my slides, to show this analogy. In aqueous veins the distribution of blood in aqueous humor is not always the same. There is not always the stratification visible which appears in this slide of the canal of Schlemm, but if the stratification is visible, we may observe changes in the ratio of blood-aqueous veins, too.

(Slide) This is the aqueous vein (indicating). Here clear fluid comes out of the region, and here it is added to the blood which is present in the conductive vein. A similar picture is here. You always see the stratification, which is not dependent on gravity, because you see, for instance here, the blood in the upper and the aqueous in the lower half.

In other words, I want to draw your attention to this analogy, this parallel. I think that we have to consider the physiological unity of this whole system of the canal of Schlemm with the so-called collectors. Blood and aqueous humor are present or not, may be present, may be mixed, in the canal of Schlemm in the so-called collectors — and a name I don't like to use is "canal."

One more word about the color of the blood seen by Dr. Kronfeld in the canal of Schlemm. I think the fact that the blood appears to be bright red does not confirm the arterial origin of this blood in the conjunctiva. You see blood of a very bright color, but that does not mean that all these vessels with bright red blood are arteries. I won't enter a discussion of why this blood shows this bright red color. It might be something in connection with the arterialization of this blood in the conjunctival epithelium through the mesoderm. Anyhow the fact that the spaces are bright red and the presence of bright red blood in the canal of Schlemm does not prove the origin of this blood, it is quite possible and fairly probable that this blood comes from the afferent arterioles.

FREDERICK H. VERHOEFF, M.D., Boston, Mass.: I should like to ask Dr.

Kronfeld whether the patients knew that these were experiments in the interest of science and that there was a certain risk connected with the experiment. In a good many of the patients upon whom he operated apparently the eyes were normal.

I would also like to ask him whether he has had one of these experiments performed upon his own eyes.

PETER C. KRONFELD, M.D., Chicago, Ill.: I greatly appreciate Dr. Troncoso's remarks. I would like only to state again that the presence of leakage in some of our cases from the canal into the trabeculum and into the anterior chamber should not be construed as evidence of pre-existing open connections between the canal of Schlemm and the anterior chamber. The leakage after the compression experiment occurs only if the pressure during the compression has been very high. After the partial anterior chamber puncture, the leakage occurs very infrequently, certainly in less than 20 per cent. After the complete anterior chamber puncture, the leakage occurs in approximately 20 per cent, which would seem to indicate that the leakage occurs more often if the actual trauma to the eye is greater.

Therefore, I would be inclined to think that these experiments confirm the presence of a very delicate wall, a very delicate separation between the canal of Schlemm and the anterior chamber, and that this delicate wall may break down under the influence of certain traumas. The greater the trauma, the greater is the likelihood of a break-down.

It is true that this method can be used to find out whether the canal of Schlemm is present in a certain eye. I interpreted Dr. Otto Barkan's last paper on the treatment of hydrophthalmos in that direction. I believe that he could actually see the red band behind the trabeculum and, therefore, demonstrated or proved to himself that a canal of Schlemm was present in those eyes.

With regard to Dr. Friedenwald's comment, Dr. Friedenwald was kind enough to tell me about his experiments with Dr. Sholz, and they are mentioned here in this paper. I am, of course, glad to know that they confirm our results.

With regard to his question whether we had performed repeated anterior chamber punctures on some of the cases with sufficiently long intervals between the two punctures to allow for complete restoration of normal conditions, the longest interval that we have allowed to elapse between two punctures was six weeks.

With regard to Dr. Ascher's remark, his idea that the picture that we showed here may represent stratification, that the entire canal may not be filled with blood, that there may be a lower layer of blood and an upper layer of aqueous, is very interesting, and I hadn't thought of that possibility. There is, however, one thought that comes to my mind. If the canal is filled immediately after compression and you watch that phenomenon of filling while it takes place, the blood simply rushes in, and it would be very difficult to conceive that during this very sudden entrance, as the blood really gushes into that canal, only a portion of the canal should become filled. The width of the area that becomes filled under those conditions is the same as that after partial anterior puncture in most cases. Therefore, just on the basis of these observations, I am inclined to think that these pictures represent complete filling of the canal, and not stratification.

With regard to Dr. Verhoeff's questions, the eyes were not normal. As I stated in the paper, there were clinical conditions of amblyopia, senile cataract, senile macula, degenerations and optic atrophies, and so on, and the patients were told that this was an experimental procedure which might for short periods improve the blood circulation to the eye.

With regard to Dr. Verhoeff's question whether any of the authors of this paper had ever undergone the procedure, I am glad to say that two of the authors had the procedure of compression of the eyeball performed on their own eyes just to find out how it felt. However, gonioscopic observations were not made. I personally have gone through the procedure of compression of the eyeball with the ophthalmodynamometer, as has also one of the other four. The procedure is slightly painful.

DR. VERHOEFF: Including the puncture of the anterior chamber that you talk about?

DR. KRONFELD: No.

SOME OBSERVATIONS AND EXPERIMENTAL STUDIES ON THE PHYSIOLOGY OF THE CILIARY MUSCLE

Erich Sachs, M.D., Detroit, Michigan

The behavior of the ciliary muscle under faradic stimulation has been observed in excised eyes (cat, dog). A phenomenon never before reported was found, and confirmed by observations of pins thrust radially through the anterior eyeball wall and of chalk particles dusted on the ciliary body through a scleral window: as the muscle contracts, the posterior ciliary-body tissue stretches forward, but the anterior coronal region expands backward, with a narrow motionless zone separating the two moving regions.

The amplitude, speed, and force of contraction at various points along a meridian were measured with micrometer scale, stopwatch, and an electromagnetic ductor calibrated in milligrams. Except for a fundal area subtending about 50° of eyeball circumference, there is demonstrable forward movement of the choroid along all meridians. Within the non-moving area of the choroid, no increase in circumferential tension within that tissue, during contraction of the ciliary muscle, could be demonstrated.

DISCUSSION

FREDERICK H. VERHOEFF, M.D., Boston, Mass.: How about the question of sympathetic stimulation? Some muscles respond to sympathetic stimulation, and others respond to nerve stimulation, to sporadic stimulation. You get them both, don't you, which confuses the results?

DR. SACHS: That is a direct stimulus applied to the muscle itself in the enucleated eye.

DR. VERHOEFF: That would take all the muscles, then. I mean suppose some muscles normally were stimulated by the sympathetic and others were stimulated by the third nerve, and in the accommodation, only those by the third nerve. In your experiments you stimulate all of them at once?

DR. SACHS: Yes.

DR. VERHOEFF: Of course, if that is true, then your results would be difficult of application to any theory of accommodation.

DR. SACHS: The same experiments have been performed by ganglionic stimulation. The first ones who observed this used ganglionic stimulation. They never observed the backward movement. As far as I can see from the experiments, they apparently put the pins just in the neutral zone or they were not immediately behind the limbus in this region. Not all eyes showed the backward movement. They had not used very many eyes.

DR. VERHOEFF: You haven't tried stimulating the nerve in the lymphatic area or even very quickly after the eye was removed?

DR. SACHS: Not yet, but we will do so. The experiments certainly have to be performed also by way of ganglionic stimulation, which will be the next task.

DR. VERHOEFF: Do you think, in your study of the muscles, that there are two sets of fibers: fibers enervated only by the third nerve, and fibers enervated only by the sympathetic? Do you think that is so?

DR. SACHS: That is a very difficult question. Personally, I do not think so. I think that just as in the iris the sphincter is relaxed by some sympathomimetic drugs which have a double action, a sympathetic action, according to the tissue, the same takes place in the ciliary muscle. I have to confess that all pharmacological experiments have failed completely. With the exception of dry nicotine, as far as I know, nobody ever got any results by using drugs in the ciliary muscle. I don't know the reason. I dropped it on the surface, I injected it into the vitreous, and only once I got a very, very slight contraction, which may have been due to the change in pH. None of the authors ever considered change of pH, that is authors of the last century.

So I can't say anything about drug stimulation, especially why drug stimulation fails absolutely on the exposed ciliary muscle — not even the strongest drugs in potent combinations.

DR. VERHOEFF: It doesn't make any difference on which side you apply it?

DR. SACHS: From the inside or the outside, or injected into the vitreous immediately below, we didn't get any reaction. They were observed for fifteen or twenty minutes. By means of this chart

method, I would have seen even very slight movements of 76 microns and less.

FRANCIS H. ADLER, M.D., Philadelphia, Pa.: Did you get any dilatation or contraction of the pupil when you stimulated it?

DR. SACHS: Very definitely: in cats, dilatation; in dogs, contraction. They react oppositely.

DR. VERHOEFF: In round pupils have you found exactly uniform reaction?

DR. SACHS: This was not a round pupil. The stimulation is a local one, and the dog's pupil always becomes oval-shaped. The dog is very peculiar, because the stimulation should dilate the pupil.

DR. VERHOEFF: Can you harmonize your theory with the Heerfordt theory of accommodation?

DR. SACHS: Yes, absolutely I can. As soon as I assume that this backward movement has nothing to do with the relaxation of the main fibers, I cannot see any mechanical influence originating from this backward movement.

DR. VERHOEFF: If that is the only kind of movement you get, how do you get the zonule relaxed?

DR. SACHS: I don't get only the backward movement. The backward movement extends over the coronoid part of the ciliary body, then the neutral zone, and from there to the larger part of the ciliary body, and the coronoid moves forward in the usual, customary and well-known way. I would assume, certainly, that is the main working part. So it doesn't contradict them, not at all, because relaxation of the zonule fibers is an essential part. Whether this backward movement influences the posterior zonule fibers or the zonule fibers which move more in the direction of the most anterior part of the ciliary muscle, I don't know. That may be, although it is very difficult to see through all the very complicated mechanical defects. I can't tell anything about it. Until I can see the primates, I won't dare to advance any theory.

SULFADIAZINE IONTOPHORESIS IN PYOCYANEUS INFECTION OF RABBIT CORNEA

Ludwig von Sallmann, M.D.
New York, N. Y.

The concentrations of the newer sulfa drugs (sulfapyridine, sulfathiazole, sulfadiazine) which enter cornea and aqueous by local administration can be considerably increased by the iontophoretic application of their sodium salts. J. Boyd demonstrated such an increase using sodium sulfathizole, and our experiments with sodium sulfapyridine and sulfadiazine give similar results.

On the basis of these findings iontophoretic treatment of the most invasive corneal infection (B. pyocyaneus) of the rabbit was initiated. The progression of the keratitis is checked in approximately half of the experiments 18 hours after inoculation, using sulfadiazine iontophoretically alone. Good results are obtained in most instances when local treatment is combined with the feeding of comparatively small doses of the drug. The combined treatment also overcomes the progression 24 hours after inoculation. The effect of the iontophoretic application is definitely superior to that of the local use of sulfadiazine powder. No damage to the corneal epithelium due to iontophoresis can be observed.

THE AQUEOUS VEINS: PHYSIOLOGICAL IMPORTANCE OF THE VISIBLE ELIMINATION OF THE INTRAOCULAR FLUID

K. W. Ascher, M.D., Cincinnati, Ohio

Aqueous veins are blood-vessel like pathways that are biomicroscopically visible. They contain a clear fluid or diluted blood and are intercalated, probably via Schlemm's canal, between the intraocular fluid on one side, and conjunctival and subconjunctival veins on the other.

Priority may be claimed for the observation of the entrance and presence of intraocular fluid in circulating blood. The fact of a continuous outflow of intraocular fluid, according to Leber's theory, was not obviously evident prior to the knowledge of these visible connections.

Only one counterpart of this fluid elimination more or less mixed with blood seems to exist in nature. Every contraction of the posterior lymph heart of the frog forces clear fluid into the renal vessels.

The anatomic substrata for the aqueous veins are the anastomosing intrascleral vessels which, according to Friedenwald, occasionally send branches to the episcleral vessels at the limbus. Therefore,

the origin of aqueous veins is found in or near the limbus often with a U like arch, one limb of which emerges out of the limbal tissue. On considering the different figures available in the literature for the amount and size of the venous outlets of Schlemm's canal and for the speed of movement in conjunctival veins we arrive at an approximate agreement between the fluid volume possibly eliminated by aqueous veins and the amount suggested by Friedenwald and Pierce.

Aqueous veins are to be recognized by their origin, course, and emptying; by their content of diluted blood or even colorless fluid; and by the response to compression of the recipient vessel. Stratification is sometimes present and will disappear on slight compression of the recipient vein. Either of two phenomena then may occur: 1) red blood cells may be forced back into the aqueous vein, in a direction opposite to the original current, or 2) the red cells are expelled out of the recipient vessel until the whole structure looks like a colorless glass rod imbedded in porcelain: the glass-rod phenomenon (GRP).

Stratification paralleling the vessel walls has been observed in the mesenterium and in the tongue of the frog but the explanation of this striation is quite a different one.

In the Cincinnati Eye Clinic, no patient with primary compensated glaucoma showed the GRP but the number of eyes investigated so far is too small for any decisive conclusion. In some eyes with secondary glaucoma, the GRP was present.

In aqueous veins, in their tributaries, and in their recipient vessels, borderlines paralleling the vessel diameter and separating sanguineous from colorless fluid may be formed. Rhythmic pulsation may occur in this borderline. The expulsion of red blood corpuscles takes place synchroneously with the acme of the radial pulse.

Sometimes a rhythmic influx of red blood cell waves ordinarily arrive in the intervals between two pulse beats.

Respiration may produce changes in the red blood corpuscle content. Lid movements, changes between light and dark as well as miotics and mydriatics and other drugs may influence the content of an aqueous vein.

DISCUSSION

FREDERICK H. VERHOEFF, M.D., Boston, Mass.: I would like to ask Dr. Ascher whether there is any significance in the appearance of these veins in glaucomatous and normal eyes, and in different types of glaucoma, too, for instance in acute glaucoma without congestion, and in simple chronic glaucoma; and whether the veins look just the same as in normal eyes.

DR. ASCHER: The number of glaucomatous eyes is not large enough from which to draw clear and complete conclusions, but we can say that the occurrence of aqueous veins is not lower in glaucomatous eyes than it is in normal eyes, or, I had better say, in eyes without intraocular pressure (abnormal intraocular pressure). However, we can see that any congestion, whether it be due to glaucoma or any kind of inflammation, interferes with glaucoma. Any trauma, puncture of the chamber, contusion, keratitis, or anything which tends to increase the red cells in the conjunctival vessels, conceals the appearance of these clear vessels, and that may be in accordance with the observations of Dr. Kronfeld this morning that when you puncture the eye, part or all of the aqueous humor escapes, and then a large amount of blood will suddenly enter the canal of Schlemm the same way the aqueous enters, and no more stratification can be possible because stratification is due only to a continuous difference of these layers. When the pressure is changed, in a moment all is destroyed.

DR. VERHOEFF: But if you had an infiltration angle and high blood pressure, the aqueous veins would be distended and the clear stream would be longer, wouldn't it?

DR. ASCHER: I don't know.

DR. VERHOEFF: You haven't observed any such thing?

DR. ASCHER: We have, but it is not clear. Our observations have not been extensive enough to make a definite answer.

ILLINOIS

The Chicago Ophthalmological Society held its opening meeting of the year at the Medinah Club, October 19. Dr. James

O. Lebensohn presented a paper entitled "The Problem of Rating Ametropic Visual Acuity," wihch was discussed by Dr. Peter Kronfeld. Dr. Sanford R. Gifford presented a paper entitled "Surgery of the Obliques," which was discussed by Dr. Samuel Meyer.

NEW YORK

The Section of Ophthalmology of the New York Academy of Medicine held its first meeting of the year in conjunction with the New York Academy of Medicine, at which time the subject "War Gases" was discussed.

Dr. Daniel B. Kirby is chairman of the section, and Dr. R. Townley Paton is secretary.

The section's next meeting will be held on November 16, at which time the instruction hour will be taken over by Dr. Edward Hartman (by invitation). He will lecture on "War Injuries of the Eye."

The following cases will be presented: Bilateral Metastatic Carcinoma, Dr. Arthur Bedell — discussion, Dr. Arnold Knapp; Neuro-dermatitis with Cataract, Dr. Clyde McDannald — discussion, Dr. Trueman Boyes.

Dr. Willis Knighton will present a paper entitled "Immediate and Late Reoperation for Glaucoma," and Dr. Brittain Payne will present one on "Fate of Eyes after Unsuccessful Operation for Glaucoma," which will be discussed by Dr. Algernon B. Reese.

The New York Society for Clinical Ophthalmology held its fifty-first regular meeting on October 5, in Squibb Hall. Dr. Adolph Posner presented an exhibit and demonstration on "Standardization of the Schiotz Tonometer." The scientific papers read were as follows: "Theories as to the Causation of Exophthalmus in Hyperthyroidism," Dr. David Marine; "Disassociation of Thyrotoxicosis and Ophthalmopathy in Graves Disease," Dr. Saul Hertz; "Surgical Procedures for Exophthalmus in Hyperthyroidism," Dr. Edmund B. Spaeth, and "Orbital Decompression in Malignant Exophthalmus," Dr. James L. Poppen. Dr. Hertz' paper was discussed by Dr. Maurice Bruger, and Dr. Poppen's paper by Dr. John H. Dunnington.

The Bronx Otolaryngological Society, Inc., met on September 22 at which time Dr. Josph F. LaSala presented a paper entitled "Facial Paralysis," which was discussed by Dr. Samuel Kulkin.

PENNSYLVANIA

The Section on Ophthalmology of the College of Physicians of Philadelphia met on October 15 in the College of Physicians Building. The scientific program was as follows: "Posterior Phlyctenular Keratitis," Dr. Glen G. Gibson — discussion by Dr. George P. Meyer; "The Etiologic Role of Focal Infection in Diseases of the Eye," Dr. Meyer Solis-Cohen (by invitation); "Classification of Congenital Ptosis," Dr. Edmund B. Spaeth — discussion by Dr. Walter I. Lillie.

THE AUTONOMIC NERVOUS SYSTEM IN HEALTH AND DISEASE

MARVIN FISHER JONES, M.D.
NEW YORK

PERHAPS it is permissible to present before this scientific assembly a paper which is somewhat of a fable, since, out of fabulous conceptions of the past, realities often have developed. These realities sometimes far surpassed the dreams. Facts of today are the realization of fantasies of the past and fantasies of today can be facts of tomorrow.

Comparatively few doctors have been interested in the autonomic nervous system, yet specialists often observe symptoms which are due to disturbed autonomic function. Many of these symptoms, which may be both subjective and objective, have not been classified under the proper causes. A more intimate knowledge of the autonomic nervous system will amply reward those doctors who are sufficiently curious to investigate. Little more can be done in the time allotted for this presentation than to indicate some of the possibilities, mention one or two practical applications and note some references which will help those who wish further knowledge.

There has been a great deal of confusion regarding nomenclature. The words involuntary, vegetative, visceral, ganglionic, splanchnic, autonomic, sympathetic and parasympathetic have been used to designate this vital nervous system. Lack of universal specific designation has added confusion. The complexity of this nervous mechanism is increasing as the ages change the vital requirements. Vital functions are controlled by this little known and less understood inter-related network of nerves.

As defined in Webster, vital functions are understood to mean, "Those functions or actions of the body upon which life is directly dependent, such as circulation of the blood, digestion, etc." There is a fairly universal acceptance of the particular vital functions which come under the influence or control of this special nervous system — the cardiovascular system, respiratory mechanism, gastro-intestinal or digestive organs, heat-regulating apparatus, and regulatory control of compensatory reactions to emotional instability.

The *vital nervous system* seems to be the proper phrase applicable to these nerve formations. Fulton in his book, "Physiology of the Nervous System" says, "The vital nervous system is understood to be cerebrospinal nuclei and tracts with widely distributed sensory and motor nerves, ganglia and plexuses which subserve vegetative functions of the body, in its peripheral ramifications. The system is characterized by synapses in ganglia lying outside the central nervous system. The motor portion is constituted by the sympathetic (thoracolumbar) and parasympathetic (craniosacral). The parasympathetic division has three primary levels of outflow, i.e., cranial, bulbar and sacral." This seems concise and understandable.

Emotional responses which have been altered in the higher plane of living still exhibit traces of days when life was reduced to fundamentals. The pilomotor reactions which produce erection of the hair on animals may be instigated by fear or anger. The heat regulating mechanism, acuity and adaptability of vision

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are activities concerned in self-protection which are controlled by the vital nervous system. With our reversion to animalism in recent years, these same elemental reflexes are again called upon to assume a major role in saving our lives.

Salivation is really the first step of a process which prepares the potential body fuel for proper assimilation. Cessation of the flow of saliva may be a result of emotionalism. A good example is the dry mouth experienced by speakers when addressing an audience. The ever-present pitcher of water seen on the speakers' tables is not primarily to replenish body fluids.

Elimination of waste materials such as perspiration, urine and feces is also a vital function controlled by the autonomic nervous system.

Further thought will call to mind many other examples which illustrate the aptness of the name *vital nervous system*. As a matter of interest, Eustachio, in 1552, first described and made fine plates of the vital nervous system.

The nomenclature from this point on will be orthodox. The name *autonomic nervous system* will refer to the system as a whole. *Parasympathetic* will be the name used to designate the craniosacral part and the name *sympathetic* will designate the thoracolumbar nervous system.

THE EFFECT OF DRUGS ON THE AUTONOMIC NERVOUS SYSTEM

Adrenalin in the blood stimulates the sympathetic nerves. Adrenalin stimulates the sympathetic system resulting in increased flow of adrenalin. Pilocarpine, muscarine, prostigmin and cholin produce effects similar to those produced by stimulating the parasympathetic nerves.

Nicotine paralyzes the synaptic connection of the preganglionic and postganglionic neurons, preventing conduction through the neuron junctions. Both the sympathetic and parasympathetic systems are similarly effected. Minimal doses block sympathetic action but with increasing doses, there is a secondary increase in stimulation. A massive dose causes paralysis which may be followed by permanently lost function.

Ergotoxine and ergotamine paralyze the terminal structure of the sympathetic motor fibers, and atropine has the same effect on the terminations of the parasympathetic fibers.

These statements regarding the effect of drugs on the autonomic nervous system seem rather academic and definite. As a matter of fact the reaction of the autonomic nervous system to these drugs is, in many instances, paradoxical. When used in individuals having different susceptibility, these drugs seem to have contradictory responses. For example, an asthmatic patient with low blood pressure may have an attack of asthma precipitated by the use of adrenalin. The usual action of adrenalin is to give immediate relief of asthma.

Fulton uses the symptoms in a syndrome to indicate the action of the sympathetic innervation of the head. He refers to Horner's syndrome for illustration. Ptosis, enophthalmos, myosis (with light reaction maintained), and the face red, hot and dry, is the picture of sympathetic paralysis. When the stellate ganglion is injected as a treatment of causalgia, the main sign for determining whether or not the ganglion has been located is the appearance of Horner's syndrome. Obviously, the reverse holds true, i.e., when the sympathetic is stimulated there is a widening of the palpebral fissure, dilation of the pupils, constriction of the skin vessels, sweating of the skin and exophthalmos.

The cervical sympathetic also supplies lacrimal, salivary and sebaceous glands in the head.

Vasomotor fibers are distributed to the entire head, mucus membrane, skin,

brain, muscles and special structures such as the eye. In this statement is much food for thought, since severance of the sympathetic nerve produces vasodilation in the mucus membrane and dryness of the surface while stimulation brings increased moisture with vasoconstriction. Stimulation of the sympathetic produces a marked and profuse secretion from the glands in the mucosa while paralysis of the sympathetic stops all impulses to the mucus glands and there is a subsequent dryness.

It would seem as though a clinical idea might be interjected at this point. Cigarette smoking certainly has an effect on nasopharyngeal and tracheobronchial secretion. When asked why cigarettes have a greater effect than cigars or pipes, the answer could be, the increased contact with an absorptive surface made possible by the act of inhaling. A higher percentage of nicotine gains access to the system and causes a greater drug action.

The phrase "smokers' cough" is no idle expression. I have tried the following clinical test with encouraging results. The dry reflex cough and the thick tenacious postnasal morning collection can definitely be improved by having the patient stop smoking for 6 weeks. This does not mean that many other things will not produce a cessation of these same symptoms nor is the statement restricted to any one brand of cigarettes. Drugs (such as iodine), changes in climate, sinus treatments and local medications may give similar relief but the cause must be removed if patients are to stay cured. There are also other causes for the symptoms of coughing and postnasal discharge so, here, as in all medicine, the cure is only effective when the diagnosis is correct.

The parasympathetic nervous system is theoretically opposite to the sympathetic in function.

"If one anterior nerve root of a spinal nerve is stimulated, diffuse sweating, pilomotor response and vasoconstriction occur over the entire trunk. Stimulation of the posterior nerve root causes a band of discreet vasodilation and inhibition of sweat corresponding to the dermatome innervated by the posterior root. It is, therefore, evident that the sympathetic division discharges en masse while the parasympathetic discharges discreetly" (Fulton). The parasympathetic appears to form a protection and restoration of vital resources as well as having a conservation function. Ganglia which lie in close relation to the ends of the nerves in the viscera are almost all parasympathetic. They possess the power of an independent short-circuit reflex.

This sympathetic-parasympathetic balance can be easily upset. It has been contended that the sympathetic system contains both efferent and afferent fibers. There is a little less assurance that afferent fibers exist in the parasympathetic. The dominant system is the sympathetic. Therefore, stimulation would exaggerate the sympathetic functions and a paralysis would not only stop its function but cause the parasympathetic system to become dominant. Drugs, disease, grandular dysfunction, dehabilitation, trauma, physical changes and emotional unbalance cause stimulation, depression or paralysis. The results of these disturbances, which may be acute or chronic, are manifested in disorders observable by the eye, ear, nose and throat specialists.

Adrenalin, atropine, nicotine and ergotamine are among the drugs which produce changes in the head through action on the autonomic system.

The two words *vagotonic* and *sympatheticotonic* deserve some attention. If adrenalin produces a strong sympathetic reaction, pilocarpine will not stimulate nor atropine paralyze the parasympa-

thetic nerves. Patients who have functional imbalance under this class are sympatheticotonic.

In persons whom pilocarpine or atropine produce a strong parasympathetic reaction, adrenalin does not affect the sympathetic nerves. Such persons are known as vagotonic.

SYMPATHETICOTONIC INDIVIDUALS AND ALLERGY

The hydrogen-ion concentration and the potassium-calcium ratio play an important role in the functional balance of the autonomic nervous system. Water elimination, metabolism and trophic regulations are affected by an unbalance. All may be disturbed in the allergic individual.

Animals deprived of their sympathetic nerves are extremely sensitive to heat and cold. They lose their normal heat rapidly. Allergic individuals, who have presumably a disordered autonomic system, are likewise sensitive to sudden temperature changes, draughts, and have attacks of hydrosis. The administration of adrenalin, in acute situations, has an almost immediate effect in bringing the allergic individual back to normal. The adrenalin in this instance supplies the temporarily lost function of the sympathetic.

When exposed to high external temperatures, animals which have been deprived of their sympathetic nerves exhibit greater rise in body temperature and marked reaction to the heat. This is evidenced by panting, increased respiration and increased salivation.

The end organs through which the nervous regulation of body temperature is brought about are mainly blood vessels. Sweat glands and internal organs whose metabolic processes tend to increase or inhibit heat production are adjuncts. The endocrine glands are also responsible for a part of heat regulation and may act by direct stimulation of the temperature center in the corpus striatum or the center in the diencephalon. It is rather to be expected that the glands of internal secretion should be functionally inter-related with the autonomic nervous system. Adrenalin is the classic example of an internal secretion which stimulates sympathetic action. Another example is found in hypo or hyper function of the thyroid gland which respectively depresses or increases body temperature. Dysfunction of the hypophysis causes disturbances in body temperature. Removal causes a fall in temperature.

EMOTIONAL DISTURBANCES

The blood picture, metabolic rate, digestive functions—in fact all vital functions may be affected by emotion. The normal body was made to meet emergencies. The body has been equipped with equalizing machinery which responds either to sudden or protracted demands. During the days when opening the front door of a home might expose a man to a missile from a hidden enemy, an attack by a hungry animal or the extended hand of a friend, the human body quickly adjusted itself to meet each situation adequately.

Security is described in the Oxford pocket dictionary as "over-confidence." The word "security" implies that the individual's right to develop and use his peculiarly human nervous mechanism in defense or attack is denied him. A human being has the right under the implications of the word "liberty" to have his emotions stimulated so that he may learn to react adequately in order to solve his own problems. Our autonomic nervous system with its associated structures was installed and has developed to be sufficient in any emergency. We like to have our personal ability challenged and in the main our response is more than enough. Our imagination is not stimulated by having our pedigree indicated on cards which

are then pulled out like puppets on a string to dangle as spineless automatons. Those persons whose autonomic systems need the soporific circumlocution talks of a salesman of ease will do well under a protectorate. Each normal person has the God-given right to encounter shocks which stimulate his autonomic nervous system. Give us courage, fear, love, hate, hunger, heat and cold so that we may be trained to respond with increasing ability, solving our problems satisfactorily. We have the power to insure our own social security and take adequate care of the health of those few unfortunates who are not so blessed. There are always those who are in themselves sufficient, but have been denied the stimulation of normal energizing emotions and therefore must whine for security. As substantiation for this pardonable generalization the following facts are submitted.

Emotion increases adrenalin output. Adrenalin exerts an influence on the resistance of voluntary muscle to fatigue. Muscular activity increases the output of adrenalin. The cycle is obvious and deduction is simple. More labor for body and mind is needed but less arm-chair philosophy.

The greater the emergency the more the sympathetico-adrenal mechanism is utilized. This very clearly states that the more you are called upon to do, if you respond effectively, the more you will be capable of doing.

Under ordinary conditions adrenalin and calcium serve as a sympathetic sedative. Under stimulating conditions the elevating effect comes into play.

OTHER ENDOCRINE ACTION

Thyroid and parathyroid secretions may sensitize the sympathetic, making it more susceptible to the action of adrenalin and nicotine. The effect of the pancreatic secretion is on the parasympathetic nerves.

Pituitrin augments the contraction of smooth muscle; therefore, it acts in conjunction with the sympathetic as a vasoconstrictor. It also sensitizes the sympathetic to adrenalin. Insulin is a parasympathetic stimulant. The posterior pituitary is a sympathetic stimulant.

LOCAL AUTONOMIC PHENOMENA

Doctor MacLean started with me to observe the effect of stellate ganglion block on hemicrania. It so happened that the patients who were to be treated also complained of tinnitus, impaired hearing or dizziness. All effects of the ganglion block were transient. However, some surprising results were obtained. Pain was relieved, but not regularly. Dizziness was aggravated in some and stopped in others. Tinnitus was affected the same way, but two patients immediately volunteered the information that they could hear better.

The anatomical arrangement of the blood supply and its sympathetic control is significant. The sympathetic system forming a plexus around the vertebral artery arises from the stellate ganglion. The fibers have been traced along the basilar artery which is formed by a junction of the two vertebrals. The internal auditory artery furnishes the sole blood supply to the inner ear and this artery is a branch of the basilar. Fibers from the sympathetic have not been traced to its ultimate distribution, but judging by other similar examples the vascular control in the inner ear is sympathetic.

In all the patients who had their stellate ganglion injected the appearance of the Horner's syndrome was considered proof of the fact that the ganglion had been blocked and in all patients who showed the Horner's syndrome some complaint relating to the ear was altered. The series was of insufficient number to form any conclusion. Unfortunately, Doctor MacLean joined

the Navy before the work could be completed. The occurrences still remain significant.

The Air Corps has been concerned with a pilot's adjustment to quick pressure changes. This adjustment is largely dependent on quick vascular adaptation plus oxygen supply. It would seem wise to have recruits examined on the basis of vagotonia or sympatheticotonia. Those with subnormal sympathetic response have difficulty in adjusting to the extreme changes in temperature encountered in flying. Their body temperature will rise in extreme heat which increases their metabolism and oxygen consumption; with a lowered oxygen supply they become apathetic. In the colder temperature they become chilled and mentally indolent. These people do not tolerate an atmosphere which is poor in oxygen or are they adaptable to a limited oxygen intake. They collapse early in an oxygen-poor atmosphere. Their movements are accomplished with greatly increased exertion and they become easily fatigued and listless. Emotional reaction to danger, opportunity or self-protection seems lost.

Hypotension in the carotid artery stimulates the sympathetic plexus or carotid sinus and there follows a consequent rise in blood pressure. A patient, on whom I did a resection of the carotid plexus, demonstrated this point perfectly. Any occluding tension on the plexus itself caused a lowering of blood pressure. Relaxing the tension would increase the blood pressure.

Any constriction around the neck of an individual with a hypersensitive carotid sinus will cause attacks of syncope. To carry this reasoning a little farther, it is not inconceivable that unusual situations imposed on fliers should cause a hypersensitivity of the carotid sinus which even the unaccustomed position of the head or pressure by equipment might influence. A doctor in my own clinic relieved his attacks of carotid sinus syncope by wearing an oversized neck band on his shirt and a very large collar.

The obvious thought is, "Why is the effect so marked on blood pressure when only one side is manipulated?" To that question, I know of no logical answer. However, these results were noted by many observers and were accurate observations. It is possible that the mass reaction characteristic of the sympathetic extends bilaterally. These observations on the human being also agree with the experimental work reported on animals.

Since this subject has been presented to eye, ear, nose and throat specialists as being a subject concerned with the head, perhaps a word of explanation is in order to account for the digression. I believe time will prove the statement, "The autonomic nervous system holds the solution to more unsolved problems in head diseases than any other structure." For some time, doctors have advocated a general medical training before specializing. Now we are faced with compulsion. In order to understand the autonomic nervous system of the head it is essential that the system as a whole be studied. It is well to recall that stimulation of one part of the sympathetic system causes a discharge of impulses en masse. The parasympathetic fibers discharge discreetly. That is to say, the sympathetic causes diffuse sweating, pilomotor response and vasoconstriction over the entire body. The parasympathetic causes a band of vasodilation and inhibition of sweat.

The actual autonomic nerve supply to the specialized parts such as the ciliary muscles, the levator palpebri, lacrimal glands and salivary glands are of much less potential importance than the vasomotor nerves and those sympathetic nerves which supply glands in the upper respiratory mucus membrane. We have all thought of these structures

from an isolated nose and throat specialist's viewpoint. Now the time has come to remember that the head is part of the body. Fenton,[1] Higbee,[3] Harkness[2] and others in our specialty have repeatedly drawn attention to the autonomic nervous system as the key to many head mysteries. Albert B. Murphy in his candidate's thesis for the American Triological Society (Ann. Otol., Rhin. & Laryng., 42:166 (Mar.) 1933) was off to a good start in his paper, "The Influence of the Sympathetic Nervous System on the Internal Ear." After the positive findings by Portman and Despons which he quotes as . . . "found constantly that there was hyperexcitability of the labyrinth after administration of drugs that stimulate the sympathetic system and hypo-excitability after giving depressors" . . . he should certainly have followed the advice given in number 7 of his conclusion. He states, "We should be conservative in our deductions on the influence of the sympathetic nervous system on the inner ear, and further experimental and careful clinical observation should be reported." I can say "Amen" to that. So many simple questions remain unanswered.

EPITOME

A fable has been presented to stimulate imagination and controversy and yet not truly a fable because facts have been interjected.

The autonomic system has been proposed for productive study. The expressive name of *"vital nervous system"* has been again suggested for descriptive clarity.

Some anatomy, physiology and pharmacology have been abstracted from such books as Cunningham's, Kuntz', Fulton's and Larsell's. At least these parts are authentic.

Cigarette smoking to excess has been given a long-needed wallop. In all probability, the local throat irritation is a minor factor compared to the effect of nicotine in paralyzing the sympathetic synapses. It is through these junctions that impulses pass in order to innervate the secretory glands of the upper respiratory mucus membranes. A dry throat, a dry cough and a bird-cage taste is to be expected.

Some quotations have been included to add prestige and at the same time recognize praiseworthy workers.

Individuals vary within normal limits. In these variations, we find the sympatheticotonic and vagotonic person. The sympatheticotonic or "go-getter" is no more to be praised than the leisurely bum. Neither one can help doing what his nervous system commands.

Allergy and the pitiful "draught sensitive" person are inextricably mixed up in the endocrine-autonomic system complex. As time goes on, the "Nose and Throater" will learn the importance of the allergic mechanism.

A few published and unpublished personal observations are included which tend to reduce the embarrassment experienced in appropriating other observers' work to use in this paper.

The vital nervous system is constructed to adjust the human being to his surroundings. The world is making terrible demands on it today. In extreme heat or cold, under positive pressure deep down in the water and far up in the rarefied atmosphere miles above the earth, machines carry men. Men must therefore direct them. The airman is given special notice because his adjusting mechanism has been drastically over-taxed. It seems appropriate to *concentrate investigation* on the vital nervous system whose job it is to supply these modern fantastic demands.

Emotions make us cry, make our mouth dry, make our nostrils dilate, make our skin blanch and cause regurgitation of our food. The emotions affect the autonomic nervous system and properly demand a reaction. If we are

given more scope and release for our emotions, the trend toward becoming inadequate automatons will shift toward the development of a productive co-operative personality.

No one has the right to rob us of the good fights in life. They prevent our becoming mentally and physically torpid. Give us more insecurity, give us more problems to solve, give us more difficult situations to meet. It is right and proper that we should grow strong through the exercise of our normal physiological heritage.

We still have brains, some independence and are always willing to work.

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DISCUSSION

W. E. GROVE, M.D., Milwaukee, Wisconsin: A paper such as Dr. Jones has just presented offers a great deal of food for thought. The autonomic nervous system intrigues me, mostly I suppose, because I know so little about it. It intrigues me because I am convinced that further knowledge about the autonomic nervous system, the endocrine glands, allergic reactions and the inter-relation of the three will unlock the doors of many obscure problems which face the otolaryngologist today. For the following facts I am indebted to Goodman, Louis and Gilman, and Alfred "The Pharmacological Basis for Therapeutics," The Macmillan Co., New York.

Without going too much into detail about the intricate anatomy involved, the known details of which can be found in any good text book, let me say briefly that the nervous mechanism of man consists of the central nervous system and the peripheral nervous system: that the peripheral nervous system is divided into somatic and autonomic portions; that the somatic portion consists of motor nerves which supply the skeletal muscles and whose cells are in the anterior horn of the cord, and sensory nerves with cells in the posterior root ganglions.

The autonomic nervous system is subdivided into the sympathetic system and the parasympathetic system. The preganglionic fibers of the *sympathetic* system leave the cord in the thoracolumbar region. No sympathetic fiber leaves the cord above the level of the first thoracic vertebra. These preganglionic fibers end in cells in a chain of sympathetic ganglia which lie entirely outside of the central nervous system. The postganglionic fibers from these ganglia are widely distributed to smooth muscle, blood vessels and glands.

The parasympathetic system consists of three outflows from the mid-brain, the medulla and the sacral part of the cord.

The main differences between somatic and autonomic nerves are that the motor nerves of the autonomic system innervate all body tissues except the skeletal muscles and that the synapses of the autonomic nerves are in ganglia outside of the cerebrospinal axis, whereas the synapses of the somatic nerves are all within the central nervous system. When somatic nerves are interrupted the innervated structures are completely paralyzed, whereas smooth muscle and glands show activity independent of intact innervation.

One of the chief differences between the sympathetic and the parasympathetic nerves is that the sympathetic nerves ramify to a greater extent. A preganglionic sympathetic fiber may travel a considerable distance and pass through several ganglia of the sympathetic chain before it finally synapses with a postganglionic neurone. It also synapses with a large number of postganglionic fibers. In some of the ganglia the ratio between a preganglionic axone and the ganglionic cells is as high as one to 20 or more. In this way a diffuse discharge of impulses from the sympathetic system is possible.

The parasympathetic system, on the other hand, has its terminal ganglia close to the organs innervated. Thus it is more discrete and limited in its discharge of impulses. In some organs there is a one to one ratio between preganglionic and postganglionic fibers.

Physiologically the sympathetic and parasympathetic systems stand as antagonists. The action of one system is brought into relief by the removal or paralysis of the other. The normal integration and inter-relation of

these two systems is necessary for the proper regulation of those body functions which are involuntary and below the level of consciousness, such as respiration, circulation, digestion, body temperature, metabolism, sweating and the secretion of certain of the ductless glands.

The sympathetic system is not absolutely essential to life, and its impulse discharges are mass discharges. The parasympathetic system is essential to life and is organized for discrete and localized discharge of impulses and not mass response.

Stimulation of the sympathetic accelerates the heart, raises the blood pressure, increases the number of red cells poured into the circulation from the spleen and redistributes the blood from the skin and the splanchnic areas to the skeletal muscles. The blood sugar increases, the palpebral fissures widen, the pupils dilate and the glands increase their secretion. In a general way stimulation of the parasympathetic systems acts in just the opposite fashion.

The drugs, which act to stimulate the sympathetic system, are adrenalin, amphetamin, ephedrin, epinephrine and neosynephrin, while ergotamine depresses it. The stimulants of the parasympathetic system are mecholyl, prostigmin, neostigmin, physostigmin and pilocarpine, while atropine, scopolamine and belladonna depress it.

To anyone who has suffered from attacks of carotid sinus syncope, as I have, and who knows what a depressing feeling it produces, it is pleasing to know that these attacks can be controlled by avoiding compression of the sinus, and in severe recurrent attacks by stripping the sinus of its sympathetic plexus as Doctor Jones has demonstrated.

Certain types of vertigo can be controlled by stimulation of the parasympathetic with prostigmin. Tinnitus can occasionally be controlled and the hearing occasionally improved by prostigmin and mecholyl. In view of these occasional therapeutic successes, further study of the autonomic system, the inter-relation of its two antagonistic portions and its integration with the endocrine glands and with allergic reactions should certainly be encouraged. Doctor Jones is to be congratulated on having brought what he calls "a fable" before us.

DAVID HIGBEE, M.D., San Diego, Calif.: I so thoroughly agree with all that Doctor Jones has stated in his paper and Doctor Grove in his discussion that there is little for me to add without repetition.

Undoubtedly the autonomic nervous system is a much neglected subject. I have recently looked through the latest text books on diseases of the ear, nose, and throat and find that little attention has been given to the facts revealed by the research of the last 20 years. These are so important in the comprehension of conditions which we encounter clinically that a chapter on anatomy, physiology, and symptomatology might well be included in each of them.

There is one statement in Doctor Jones' paper that is of special interest to me. It concerns the relationship of pain to the autonomic nervous system. Pain is a phenomenon of the cerebral and thalamic centers. It can only be sensed through afferent fiber tracts. The autonomic nervous system is an effector system composed entirely of efferent fiber tracts. It conducts stimuli initiated through the organs of special sense and through the somatic and visceral components of the sensory nervous system to perform special functions such as vasal constriction and the control of glandular function.

It is fundamental in physiology that a fiber system will conduct a stimulus across a synapse in only one direction, namely, from dendrite to axon and never from axon to dendrite. How then can pain, the function of an afferent fiber tract, be relieved by the injection of a ganglion of an efferent fiber tract? One explanation may be that a vasospasm, which is capable of stimulating afferent tracts, was relieved by the injection. Another explanation may be that although the ganglion was injected as proven by the development of Horner's syndrome, yet some of the drug may have been injected outside the ganglion as well, in which case the visceral components of sensory fibers of the cervical nerves would have been blocked.

I believe that we do not relieve pain in the head by any application or injection of any drug to ganglia of the autonomic nervous system. Pain in the head is conducted to the sensorium by the fifth and seventh nerves. Fibers of the sphenopalatine ganglion are efferent fibers carrying impulses which control the circulation and glandular activity. Whenever pain is relieved by cocainization or injection, it is through the effect of the drug on the sensory fibers of the fifth or seventh nerves. If only the fibers of autonomic ganglia themselves were affected, the resultant action could only be paralysis of their role in circulation and glandular activity.

Doctor Jones has referred to the relationship between our emotions and the autonomic nervous system. In these days this reference

is timely and should be appreciated by all of us. We are accustomed to thinking of the autonomic system as one which functions below the level of consciousness and not subject to volitional control. This is not wholly true. We can exercise a great deal of control over its functions. Witness the individual who becomes calm in the face of danger and who, by repeated exposure to great hazards, loses all fear. This is a phase of building up the morale of an individual and when multiplied many times determines the mass reactions of communities which withstand adversity. Morale itself perhaps might be described as the optimum correlation of the functions of the cerebral and the autonomic nervous system.

I. G. Spiesman, M.D., Maywood, Ill.: Doctor Jones' paper is certainly very timely, and I believe we should have many more like it. I would simply like to add two more phases to it. The first consists of the vasomotor study of the nose in relation to the common head cold, the effect of the application of cold to the skin surface and contraction of the vessels of the mucous membrane of the nose, measured by a potentiometer and various selectors which have been developed for that purpose. Considerable investigation has revealed that a vasomotor change occurs in those people who have so very many colds — I am referring to the infectious head cold — and that these people simply are unable to adjust themselves to rapid changes in temperatures such as occur, for instance, in this Great Lakes city. We feel that that inability is in many instances responsible for the frequent and repeated head cold.

The other addition to the paper already read is the study of the emotional side — the emotional effect of worry, anxiety, fears of the patient and his great many colds. We have records of patients in this climate who have had as many as one cold every 6 weeks or one cold every 3 weeks, all of an infectious character. These are the all-year-around variety of colds.

Emotional upsets may be the cause for a patient's having a cold every 6 weeks throughout the spring, summer, fall and winter. When it is possible to correct the mental status of the patient, it may also result in control of the number of his colds.

In this climate everybody is entitled to one good cold a year — two is considered not unusual. However, there are patients who have 6 or 7 infectious colds per year that are not always due to the patient's emotional state; but such frequent infectious colds may be a factor in disrupting his emotional equilibrium.

THE SURGICAL MANAGEMENT OF CARCINOMA OF THE MID-THORACIC ESOPHAGUS

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BY INVITATION

THE TECHNICAL aspects of the surgical removal of carcinoma of the esophagus vary according to the location of the growth. Those arising in the neck do not concern us here. It is convenient to divide the thoracic esophagus into fourths. The upper fourth is that portion which extends from the base of the neck to the superior surface of the aortic arch. Carcinoma arises in this segment rarely and presents difficulties of management which have not yet been successfully worked out. The middle half of the thoracic esophagus reaches from the superior margin of the aortic arch to the level of the inferior pulmonary vein. A growth located in this portion necessitates the performance of a partial esophagectomy with establishment of a cervical or upper thoracic esophagostomy and a gastrostomy for purposes of alimentation. Ultimately in such cases a rubber tube connection or a skin plastic channel constructed between the upper and lower stomata makes it possible for the patient to take nourishment by mouth. The remainder of the esophagus, which extends from the inferior pulmonary vein to the cardiac orifice of the stomach, comprises the lower fourth.

It has been suggested elsewhere[1] that an arbitrary division of the thoracic esophagus and cardiac region of the stomach into 3 zones is convenient for the purpose of delineating the technical problems involved in the surgical approach to carcinoma located in this region. The upper fourth comprises Zone 1. The middle half is designated as Zone 2. The lower fourth with the abdominal segment of the esophagus and the cardiac portion of the stomach falls into Zone 3. In Zone 3 a resection followed by an esophago-gastric anastomosis can usually be carried out.[1,2] In this paper we are concerned only with carcinoma located in the middle half of the thoracic esophagus (Zone 2).

PATHOLOGICAL CONSIDERATIONS

(1) *Extension within the wall of the esophagus.* Experience has shown that carcinoma of the esophagus often extends in the submucous and muscular layers for a long distance beyond the apparent limits of the tumor. In the intramural lymphatic channels it may be found several centimeters beyond the growth. It is obvious, therefore, that any operation calculated to remove these tumors must be planned so as to remove a long segment of esophagus or even ideally the entire thoracic portion.

(2) *Distant metastases.* Metastases from carcinoma of the esophagus occur in the regional lymph nodes early in the course of its development. Lesions located in the middle half and lower fourth of the organ are very prone to invade the retroperitoneal lymph nodes below the diaphragm and show a special preference for those situated in the region of the left gastric vessels. In a series of 30 patients who were explored abdominally as a first stage before performing an esophagectomy, metastases

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were found in the retroperitoneal nodes near the left gastric vessels in 15 cases. It is important in every case to have accurate knowledge about the condition of these nodes in order to decide whether a given growth is operable or not. Because the esophageal veins communicate directly with the caval system, metastases to the lung are not infrequent. Roentgen-ray examination of the chest should therefore be included in the preliminary clinical investigation of these patients. Metastases to the liver occur late in the course of the disease.

(3) *Invasion of Adjacent Structures.* Local fixation with direct invasion of the adjacent organs and tissues has a twofold importance in cases of carcinoma of the esophagus. In the first place it often defeats the surgeon in his attempt to perform a resection. When the growth is intimately adherent to the left pulmonary vein or artery, the left main bronchus, or the arch of the aorta, it is frequently impossible to remove it. Unlike carcinoma elsewhere in the gastrointestinal tract, where portions of adjacent organs or the abdominal wall can be removed with an adherent growth, it is impossible to excise portions of any of the vital structures to which a carcinoma of the mid-thoracic esophagus is often intimately attached. Of 13 patients on whom a thoracotomy was performed with the intention of resecting a carcinoma of the esophagus, the growth was found to be locally inoperable in 5 cases. In 2 of these it was impossible to free it from the arch of the aorta. In one the left inferior pulmonary vein was invaded by the growth. In 2 others the growth was too adherent to the left main bronchus and pulmonary artery to permit resection. Invasion of the right mediastinal pleura is of little consequence. A portion of this membrane can be removed and the defect closed if possible or, if need be, the opening may be left without serious consequences.

In the second place the proximity of the esophagus to the aorta and the structures of the hilum of the left lung is of importance in the prognosis following operation. It is obvious that the dissection must always be done through tissues which are very close to the growth and therefore frequently invaded with malignant cells. Even though it is possible to remove an adherent growth, the end result is almost certain to be a recurrence because of the necessity of cutting so close to it. Of 9 patients on whom an esophagectomy was successfully carried out, 4 have died of local recurrence and metastases. In all of these the growth was so adherent as to make removal difficult. The 2 longest survivals to date are patients whose growths were small and not adherent. The 2 other living patients were operated upon too recently to know the outcome. An adherent carcinoma of the esophagus, even though it may be removable, is probably incurable. For this reason when enough experience with resection of this lesion has been accumulated so as to make comparisons of the ultimate survival rate in this disease with that of carcinoma elsewhere in the gastrointestinal tract, the result will probably be disappointing. But the hopeless outlook and the unsatisfactory results of palliative treatment make it worth while to operate in every case where possible.

PRE-OPERATIVE EVALUATION OF THE PATIENT'S CONDITION

1. *Clinical Observations.* (A) *Nutritional State.* The early development of obstruction in cases of carcinoma of the esophagus inevitably leads to loss of weight and evidences of malnutrition in all these patients. Not infrequently they are in a state of starvation when first seen, but this does not necessarily mean that the growth is inoperable.

(B) *Age and Cardiovascular Condition.* The condition of the patient's cardiovascular and renal functions must be

carefully evaluated. Advanced age in itself does not always contra-indicate esophagectomy. The operation was carried out successfully in one patient at the age of 72. This patient is now alive and well almost 2 years later.

(C) *Build and Structure of the Patient.* Of great importance is the general build and structure of the patient. The individual with a barrel-shaped chest, a short neck, and a heavy abdomen is not a good subject for this type of surgery.

(D) *Pain and Persistent Fever.* A persistent pain in the back often means that the growth is inoperable. Likewise, a persistent fever almost invariably indicates that the lesion has reached an inoperable stage. The fever results from deep ulceration and spread of infection in the peri-esophageal tissues.

2. *Esophagoscopy.* Inspection of the lesion by esophagoscopy should be carried out to confirm the diagnosis, but it is not possible to decide about the operability of a carcinoma of the esophagus by this means. The growth may extend much farther along the deep layers of the esophagus than one would anticipate from its appearance when seen from within the lumen.

3. *Roentgenography.* Roentgenologic visualization of the lesion is of the greatest value in determining the degree of extension of the growth in the esophageal wall, although the amount of fixation and peri-esophageal infiltration of the tumor cannot be accurately estimated even by this means. The demonstration by x-ray studies of an esophagotracheal fistula or the escape of barium into a sinus in the mediastinum, which indicates that the growth has ulcerated through the esophageal wall forming a localized peri-esophageal abscess, obviously excludes any hope of radical surgery.

OPERABILITY

From January 1, 1940, to September 1, 1942, a period of 2 years and 9 months, 69 patients with carcinoma of the esophagus were treated at the Massachusetts General Hospital. After excluding the obviously hopeless cases and those where an exploration was performed but the growth found inoperable it was possible to perform an esophagectomy in 10 patients from among the entire group. In this series therefore a radical surgical excision could be performed in only 14.3 per cent of the entire group originally seen. Some of the reasons for this low figure have been discussed.

SURGICAL MANAGEMENT OF CARCINOMA OF THE MIDDLE PORTION OF THE THORACIC ESOPHAGUS

Adequate removal of a carcinoma located in the middle half of the thoracic esophagus precludes the possibility of performing an anastomosis within the chest for the restoration of continuity. In all these cases it is necessary to establish a cervical esophagostomy and a gastrostomy or enterostomy for feeding. This can be done in one operation, but certain advantages result from dividing the procedure into 2 stages.

First Stage of the Operation. The first stage is carried out through a left upper quadrant rectus muscle-splitting incision. The abdomen is explored for metastases and the liver is palpated. The lymph nodes in the region of the left gastric vessels are examined and a frozen section biopsy may be done. If these nodes are extensively involved, it is doubtless unjustifiable to subject the patient to the discomforts and operative risk of esophagectomy. In such a case if the obstruction is severe, a palliative gastrostomy is then carried out. If the obstruction is only partial, more satisfactory palliative relief is obtained by bouginage with or without x-ray treatment. In the absence of metastases be-

low the diaphragm, a gastrostomy or jejunostomy is performed. The choice between these 2 types of enterostomy depends upon whether or not a plastic reconstructive procedure is contemplated and if so upon the type of procedure to be utilized. (1) A *jejunostomy* is performed if one intends to utilize the lower segment of the esophagus for the lower stoma. (2) A *permanent gastrostomy* is constructed if the lower esophageal segment is not to be used. The choice between these 2 types of stomata may be a matter of individual preference, but experience has shown that although the esophageal mucosa heals with the edges of the skin more easily than does the gastric mucous membrane, there is serious danger of a recurrence of the disease in the stoma. On the other hand, the construction of a Beck-Jianu gastrostomy makes it possible to place the lower stoma at a much higher level than the use of the lower esophageal stump unless, as in one method to be described, the fundus of the stomach is completely mobilized and brought up beneath the skin of the chest wall.

Second Stage of the Operation. After an interval of approximately 2 weeks the esophagectomy is performed by the trans-pleural route. At present there is a controversy over the relative merits of the right-sided approach as compared with that through the left side. A thorough discussion of this subject would be out of place here.[1] In brief, I consider the left side preferable because it affords better access to both the upper (supra-aortic) end and the lower portion of the thoracic esophagus. The difficulty of separating the growth from the arch of the aorta may be overcome by cutting the upper intercostal arteries and retracting the aorta forwards. At this stage of the operation the esophagus is removed either by pulling the entire growth-bearing portion up through the neck, according to the original Torek technique,[4] or by removing this portion within the chest, thus leaving the upper segment only to be withdrawn through the cervical incision. Details of the operative technique are omitted but have been carefully described elsewhere.[1] It is important to avoid division of the upper portion of the esophagus more than 4 cm. above the aortic arch because of the short proximal stump which remains. If this end is so short that the stoma must be left in the neck above the level of the clavicle, it is difficult for the patient to use a rubber tube connection. A suitable position for the proximal stoma is just below the level of the clavicle.

After adequate time has elapsed to ensure healing of the upper esophageal stoma to the skin, a rubber tube connection is fitted between the upper and lower orifices. With this the patient can usually swallow liquid and often soft solid foods. Gain in weight and strength is prompt, but the mechanical arrangement is rarely very satisfactory. A certain amount of leakage is likely to occur. Irritation of the skin often results. The care of the tube and the difficulty of holding it in place are troublesome. It is therefore desirable to construct an external esophagus by some type of plastic procedure if possible. For various reasons, such as age or poor general condition of the patient, this may be impractical. In some cases the patient is sufnciently contented with his rubber tube to refuse it.

TECHNIQUE OF CONSTRUCTING A TUBE OF SKIN FOR AN ANTETHORACIC ESOPHAGUS

Of the many methods proposed for constructing an antethoracic esophagus only those which involve the use of the skin from the anterior thoracic wall are practicable. The details of the problem inevitably vary from case to case.

(1) *Usual Technique.* The operation is performed in 2 or more stages and is

applicable for use with 3 types of lower stoma which will be discussed. Two parallel vertical incisions are made about 6 centimeters apart and extending from just below the level of the upper esophageal stoma to a level near the lower stoma. The intervening skin is then freed along each edge for a distance of approximately 2 centimeters toward the center where a similar width is left attached. The ends of the partially mobilized flap are not cut. The edges are then turned toward each other and approximated with 2 layers of fine silk sutures over a soft rubber tube of suitable diameter. This tube is removed in 7 to 10 days. The outer aspect of the skin tube and the denuded areas on each side of it remain to be covered. There are 2 ways to accomplish this: (a) In women who because of the breasts have a much more flexible skin, the two lateral edges can be mobilized and brought together over the inverted skin tube. In this way all the denuded surfaces are easily covered in one manipulation. (b) In men because of the lack of flexibility of the skin over the chest wall the 2 lateral edges can usually be brought only as far as the sides of the inverted skin tube where they are held by mattress sutures passed be-

FIG. 1 — First stage of construction of skin tube for antethoracic esophagus. Healing complete. Upper and lower ends not yet connected with stomata.

neath this tube. The 2 denuded areas on each side of the skin tube are covered in this manner. The raw surface of the skin tube itself is then covered with a large Thiersch graft taken from the patient's thigh. (Fig. 1.)

At a second stage using a similar technique, the skin is infolded over the upper stoma and the denuded area covered either by direct suture of the remaining edges or by grafts from the thigh.

As a final stage the portion of the skin tube around the lower stoma is constructed in the same manner. In cases where there is no jejunostomy and feeding must therefore be carried out through the lower stoma, a rubber catheter is inserted through a short incision placed in the side of the central portion of the skin tube and passed through the stoma into the stomach before the inversion of the lower portion of skin is completed. (Fig. 2.) After healing is complete the catheter is removed and the patient is allowed to take liquid and soft solid foods by mouth. The short lateral incision heals promptly.

(2) *Method Proposed for a Man with Hair on the Chest.* A man with a hairy chest presents a special problem. Growth of hair within an artificial esophagus made from inverted skin would result in functional difficulty. X-ray epilation has not been used because it is antici-

FIG. 2 — Completed antethoracic esophagus 1 week after final stage. Catheter for feeding still in place.

pated that the relatively large dose which is required to bring about the desired effect would interfere too much with the nutrition of the skin flap. Epilation by electrocoagulation is impracticable where there is a large amount of hair. In some cases the lower stoma can be placed in a position where a relatively hairless portion of skin can be utilized. In other cases no such area can be found. In one such case a long pedicle graft of inverted skin constructed from the side of the chest wall and covered on the outside with Thiersch grafts was utilized. Unfortunately this patient died of recurrent disease before the final stage of the plastic operation was completed.

been utilized in the construction of an antethoracic esophagus in three different ways, depending upon the type of lower stoma which is used.

(1) *Use of the Lower End of the Esophagus Brought Up to the Abdominal Wall.* (Fig. 3.) A preliminary jejunostomy having been carried out at the time of the abdominal exploration and the esophagectomy having been successfully completed, the first step towards constructing an antethoracic esophagus using the short lower esophageal segment is to re-open the upper abdomen and, after mobilizing the cardiac region of the stomach by dividing the left gastric vessels, to bring the

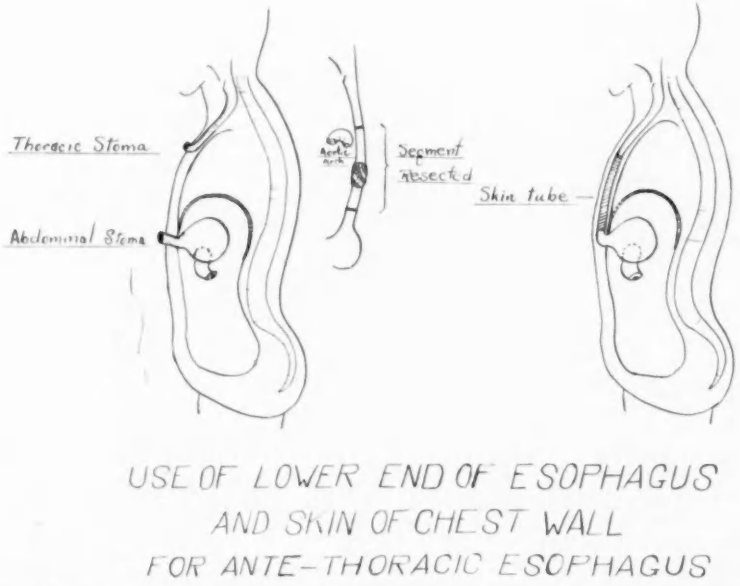


FIG. 3 — Diagram illustrating the method of constructing an antethoracic esophagus by connecting a skin plastic tube made from the chest wall with a cervical esophagostomy stoma above and an abdominal stoma made by bringing the lower esophageal segment to the skin of the upper abdomen.

METHODS OF UTILIZATION OF SKIN TUBE IN THE CONSTRUCTION OF AN ANTETHORACIC ESOPHAGUS

The above-described method of constructing a tube from the skin lying between the upper and lower stomata has

lower esophageal end to the surface of the abdominal wall. Here it is fixed in the wound. After this wound has healed, the skin plastic procedure is begun. The central portion of the skin tube in this type of case must usually be made in

two stages because of the long distance to be covered. The upper and lower ends are then completed in the usual manner. The disadvantage of this method is that such a long skin plastic tube is required and that there is likely to be difficulty in completing the lower connection because of the angulation which results from its passage over the left costal margin. In one case there was a partial stenosis at that point.

(2) *Use of the Lower End of the Esophagus Brought Halfway Up the Chest Wall by Mobilization of the Stomach.* (Fig. 4.) A better functional result can be obtained by this method which has the advantages of avoiding the difficulty of angulation at the costal margin and of requiring a much shorter skin plastic tube. This procedure is actually a modification of the first method. Instead of merely mobilizing the lower esophageal segment, the entire fundus of the stomach is freed by cutting the left gastric vessels, the vasa brevia, and the left gastro-epiploic vessels. This frees the entire proximal half of the stomach which is then drawn up over the lower portion of the chest wall in a subcutaneous channel to a point just above or near the level of the nipple where the end of the lower esophageal stump is brought through a short incision in the skin. (Figs. 5 and 6.) A patient on whom this method of constructing an antethoracic esophagus was used is living and well with excellent deglutition two years after the operation.

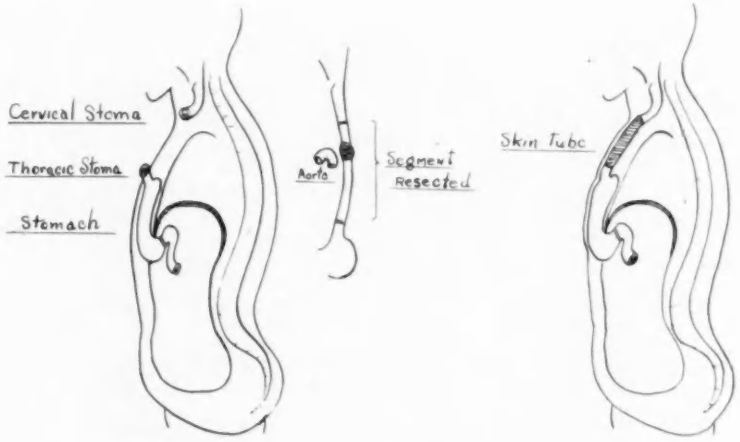


FIG. 4 — Diagram illustrating the method of constructing an antethoracic esophagus by connecting a skin plastic tube made from the chest wall with a cervical esophagostomy stoma above and a lower thoracic stoma made by bringing the lower esophageal segment and the entire fundus of the stomach up beneath the skin of the lower anterior chest wall.

FIG. 5 — Chest wall of patient after esophagectomy before constructing an antethoracic esophagus. Cervical esophagostomy stoma above left clavicle. Lower esophageal stoma medial to left nipple. Fundus of stomach lying beneath the skin of the lower chest wall shows as a bulging prominence.

FIG. 6 — Completed antethoracic esophagus 1 month after completion of final stage.

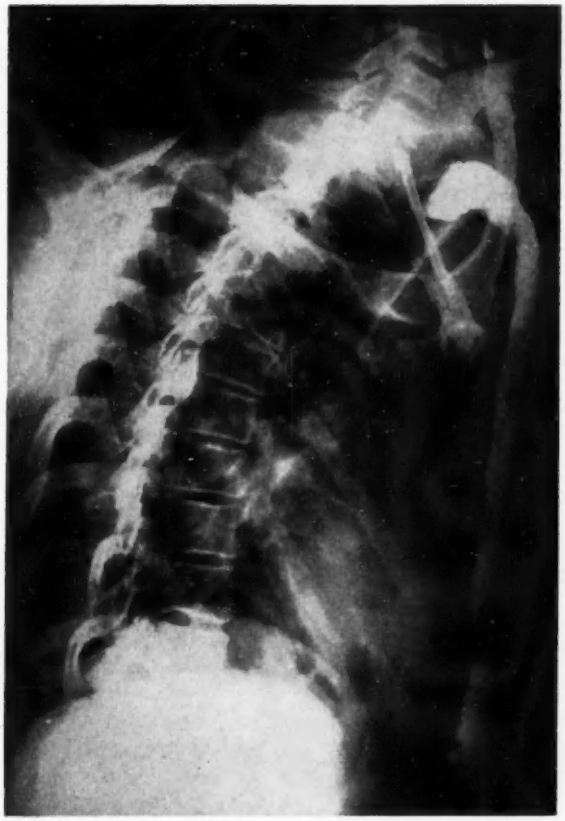
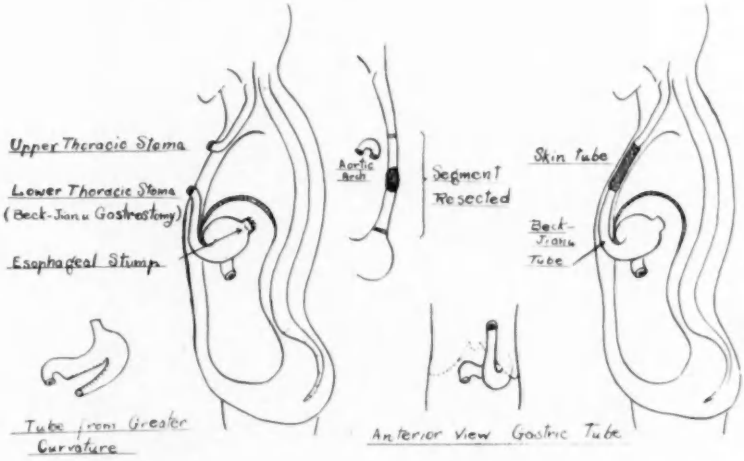


FIG. 7 — Roentgenogram of completed antethoracic esophagus taken just after patient had swallowed barium by mouth. Oblique view.

(3) *Use of a Beck-Jianu Gastrostomy for the Lower Stoma.* (Fig. 8.) A Beck-Jianu gastrostomy tube of relatively large diameter and sufficient length can usually be brought up the chest wall as far as the level of the nipple or above. Experience with this type of gastrostomy as a method of palliation in inoperable cases has been reported in a previous publication.[3] By using this method of constructing a lower stoma a relatively short skin plastic tube is required. It has the further advantage of saving one step in the multiple stage procedure because it is performed at the preliminary abdominal exploration. By its use a jejunostomy is avoided.



USE OF BECK-JIANU GASTROSTOMY FOR ANTE-THORACIC ESOPHAGUS

FIG. 8 — Diagram illustrating the method of constructing an antethoracic esophagus by connecting a skin plastic tube made from the chest wall with a cervical esophagostomy stoma above and a lower thoracic stoma made from a Beck-Jianu gastrostomy tube.

Patients on whom an antethoracic esophagus has been constructed swallow surprisingly well. Liquids pass through the inactive tube of skin with ease by the effect of gravity alone. More solid foods may be washed down with draughts of fluid, but the patient usually learns to speed the passage of such foods along by sweeping the hand down over the food-filled channel so as to milk the contents downwards into the stomach.

SUMMARY OF EXPERIENCE WITH THE SURGICAL REMOVAL OF CARCINOMA OF THE MIDPORTION OF THE THORACIC ESOPHAGUS

From February, 1940, to September, 1942, 30 patients with carcinoma of the midthoracic portion were considered suitable for resection. (Chart I.) A preliminary abdominal exploration was carried out in each case. Fifteen of these patients were excluded as candidates for esophagectomy because of the presence of lymph node metastases within the abdomen. Of the 15 patients who did not have positive nodes below the diaphragm, 2 failed to come to exploration of the chest, one because metastases were recognized in the lungs and

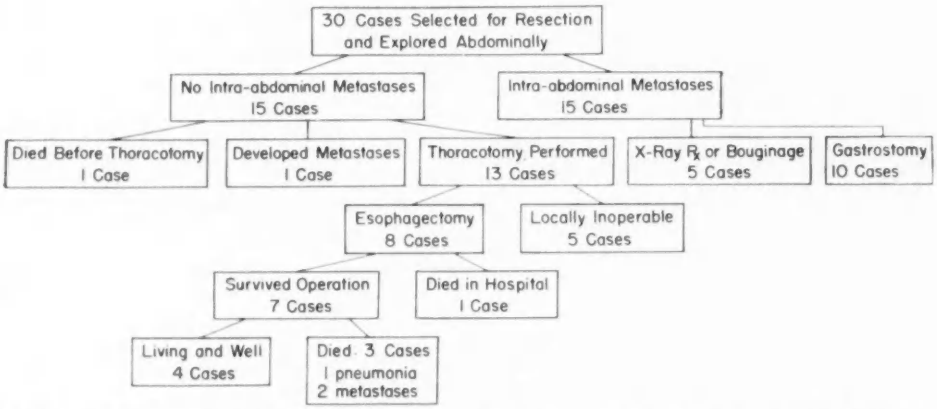

30 Cases Selected for Resection and Explored Abdominally
No Intra-abdominal Metastases 15 Cases
Intra-abdominal Metastases 15 Cases
Died Before Thoracotomy 1 Case
Developed Metastases 1 Case
Thoracotomy Performed 13 Cases
X-Ray Rx or Bouginage 5 Cases
Gastrostomy 10 Cases
Esophagectomy 8 Cases
Locally Inoperable 5 Cases
Survived Operation 7 Cases
Died in Hospital 1 Case
Living and Well 4 Cases
Died 3 Cases 1 pneumonia 2 metastases

CHART I — Fate of 30 patients with carcinoma of the midportion of the thoracic esophagus selected from a larger group as probably having a resectable growth. February, 1940, to September, 1942.

TABLE I

Esophagectomy for Carcinoma of the Midportion of the Thoracic Esophagus (Zone 2)

Name	*Date*	*Age*	*Result*
E. P.	1938	43	Died with metastases. External esophagus completed.
E. C.	1939	63	Died postoperatively.
R. B.	1940	48	Died with recurrence.
C. W.	1940	53	Died with recurrence.
A. M.	1940	59	Died with recurrence. External esophagus completed.
J. L.	1940	48	Well. External esophagus completed.
P. L.	1941	57	Died 4 months after operation.
J. G.	1941	72	Well.
B. L.	1942	68	Died postoperatively.
C. H.	1942	60	Well.
A. S.	1942	36	Well.

spine and the other because of death from pneumonia. Thirteen patients came to thoracic exploration. Of these 5 were found to have a locally inoperable growth and a resection was not attempted. A resection was carried out in 8 patients. Of these one died of pneumonia.

Of the 7 patients who survived esophagectomy, 3 have died. One of these died of pneumonia during the process of making an external esophagus. The other 2 died of local recurrence and metastases, each approximately one year after the esophagectomy. The four ultimate survivors are apparently well. Two of these were operated upon approximately 2 years ago. The other 2 have gone less than a year since operation.

Table I shows the total group of 11 cases of carcinoma of the midthoracic esophagus in which an esophagectomy was performed, including 3 cases done before February, 1940. These 3 were not included in the above analysis of cases because of the lack of investigation of the abdominal lymph nodes at the preliminary laparotomy. It will be seen from this that out of a group of 11 cases of esophagectomy there were 2 operative deaths and 9 survivals. Of the 9 patients who survived, 5 are dead, one from pneumonia and 4 because of

recurrence or metastases. Four of the original 11 patients are alive.

Although carcinoma located in the lower fourth of the thoracic esophagus (Zone 3) is not the subject of this paper, it may be of interest for the sake of completeness to state that 5 patients with an esophageal carcinoma in this location have been operated upon. In each case a resection of the lower esophagus and cardiac portion of the stomach was carried out. The operation was completed by performing an esophagogastric anastomosis. One of these patients died as a result of operation. One died of recurrent disease one year after resection. Three are alive and well.

Table 2, which includes the 2 types of cases, shows that in all, 16 patients with carcinoma of the thoracic esophagus have been treated by surgical extirpation of the growth. Of these, 3 died postoperatively. Five have died of recurrent or metastatic disease. One died of intercurrent disease (pneumonia). Seven are living and well 2 years or less after the operation.

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DISCUSSION

D. B. PHEMISTER, M.D., Chicago, (by invitation): Doctor Sweet has given you this outstanding contribution concerning his work and that of his colleagues at the Massachusetts General Hospital on this rapidly developing subject of the surgical treatment of carcinoma of the esophagus.

One thing which it demonstrates unquestionably is that when carcinoma of the esophagus is localized, it can be resected transthoracically with a relatively low mortality. Doctor Sweet had 11 cases in which the carcinoma was situated in the mid-thoracic region and in which 2 deaths occurred postoperatively. I have had 6 cases in which one death occurred postoperatively as a result of contamination and infection of the mediastinum.

Up to 6 years ago these operations were performed only occasionally, and the results were very poor. Now they are being done regularly in a number of clinics in this country, with relatively good operative results.

A relatively standardized technic has been established in performing resections for carcinoma of the lower esophagus and of the proximal end of the stomach. In a one-stage procedure a transthoracic resection is carried out and the distal end of the esophagus and the stomach are anastomosed. For cases of carcinoma located higher up in the esophagus, such as Doctor Sweet has discussed, the operative technic is decidedly in a developmental stage, because the number of surgical procedures necessary, the dangers involved because of the level of the tumor and the possibilities of approach are much greater.

I have been trying to perform these operations in one stage, doing away with the preliminary laparotomy and exploration. There

TABLE II

Total Experience with Resection of Carcinoma of the Thoracic Esophagus (Zones 2 and 3)

	No. cases	*Died post-operatively*	*Died of metastases, recurrence, or intercurrent disease*	*Living and well*
Zone 2 (middle half)	11	2	5	4
Zone 3 (lower fourth)	5	1	1	3
TOTAL	16	3	6	7

are advantages and disadvantages to that procedure. I always open the left side of the chest. I agree with Doctor Sweet that the approach is better from that side. I explore the esophagus in the vicinity of the tumor, and if the tumor appears to be resectable, then I open the diaphragm and explore the abdomen. It has been demonstrated by operations performed on the proximal end of the stomach and the distal end of the esophagus that, after opening the abdomen, a relatively satisfactory exploration can be made of the addomen for the presence of metastasis there.

Then the resection is carried out. If there is a sufficient portion of the esophagus below the level of the tumor that seems to be uninvolved, a stump is preserved. The esophagus is divided, and it along with the preserved stump below is carried forward and an inferior esophagostomy is performed through an anterior incision. The upper portion is then taken out through the neck and the superior esophagostomy established. There is objection to leaving this stump. Doctors Sweet and Churchill have reported recurrences as a result of doing so. I too have recently had that experience. Two years ago I did a resection and made an inferior esophagostomy, and the day before yesterday the patient returned with a recurrence in that stump. That is argument in favor of removing the esophagus in all cases, as Doctors Sweet and Churchill do.

Even when all the esophagus is removed from below, through the diaphragm, the greater and lesser curvatures of the stomach can be freed the same as is done when a large portion of the stomach is resected and anastomosis is performed and brought forward, establishing the inferior stoma.

It will require time in order to standardize this operative procedure, and there may be modifications beyond those which have already been developed.

There is just one more point which I would like to mention. In 3 cases I have divided the thoracic duct. In 2 cases it made no difference. In one case there was an accumulation of fluid in the upper mediastinum, but there was resolution of that exudate, without any serious consequences to the patient.

FLETCHER D. WOODWARD, M.D., Charlottesville, Va.: I would like to thank Doctor Sweet for his presentation, and to compliment him on the wonderful results obtained. It is a remarkable achievement when one can report 7 patients out of 16 alive and well after esophagectomy. Here figures are quickly stated, but I can assure you that they represent years of hard and courageous work, for a successful esophagectomy is also beset by "blood, sweat and tears."

The advance in the knowledge of cancer of the esophagus, as well as in the diagnosis and technique of its surgical removal, has been marked since Doctor Torek reported the first successful surgical removal of the esophagus in 1913. His patient lived 13 years, and subsequently died at the age of 80 from pneumonia, a result which has yet to be equalled.

Surgery offers the best hope of cure today, so it is incumbent upon us to see that earlier diagnoses are made, and to assist the thoracic surgeon in determining the operability of each case. To this end I would like to bring out a few facts which Doctor Sweet did not attempt to mention, because his paper was limited to the technic and report of cases.

Early diagnosis is most important if the high percentage of inoperable cases, due to local and regional lymph node extension, is to be lowered. To make an early diagnosis, the importance of slight and persistent dysphagia should be impressed on the minds of all physicians, for this symptom is early, constant and significant of malignancy, especially in those patients within the cancer age. The most frequent tumor of the esophagus is carcinoma.

By means of roentgenographic examination, using a contrast medium and spot films, the suspicion can be verified in most instances. Esophagoscopy will give much additional information, for if the tumor presents intra-esophageally as an ulcerative, nodular, polypoid or cauliflower-like mass, a biopsy can be taken. If, on the other hand, no mucosal changes are noted, an attempt at biopsy is unwise, because of the danger of perforation of the esophageal wall. However, the extent of fixation and degree of induration can be estimated by palpation with the tip of the esophagoscope.

Operability is also determined, as Doctor Sweet has mentioned, by roentgenographic examination of the chest, bronchoscopy, palpation of cervical and axillary lymph nodes and, if necessary, biopsy, and by abdominal exploration for metastases to the liver or regional lymph nodes. Also, the general condition, age and build of the patient must be considered. If metastases are found on abdominal exploration, a palliative gastrostomy can be done.

Surgical removal is most successful in the lower end and least successful in the upper

end of the esophagus. Doctor Sweet's suggested zoning of the esophagus will no doubt aid materially in future discussion of this problem.

Removal of carcinomata of the esophagus by electro-surgery and radiation both by radium and x-ray given by the fractional dose method has been disappointing. However, it is likely that future developments in the technic of application of radiation may bring encouragement. This development is particularly needed at this time for upper esophageal tumors. Among the more recently suggested methods of radiation therapy is the application of radium or direct application of the roentgen ray after the tumor has been exposed surgically. But, until this time comes, we must work for earlier diagnosis and depend on surgery, and I am sure that after hearing Doctor Sweet's presentation today, we are justified in considering carcinoma of the esophagus in a much more hopeful way than formerly.

HARRIS P. MOSHER, M.D., Marblehead, Mass.: I have lived a life with carcinoma of the esophagus, and it has been a very dreary and disappointing life in connection with that subject. I have never seen a cured case of carcinoma of the esophagus until I saw the cases of Doctor Sweet. Let that go for an introduction.

Let me say a word to Doctor Sweet. I am very much obliged to him for accepting the invitation to come before this society, and I take a little credit to myself in bringing his name before this society.

Now coming to the subject in hand, I feel that a new era has begun in the treatment of carcinoma of the esophagus, and I think it is a very hopeful era. I am a bit disappointed that he did not show the technic of the thoracic approach. I have tried to keep in touch with general surgery, and I know some of you men have, and some of you have not. But, if you have not and you want one of the experiences of your medical life, see the thoracic approach to the esophagus.

E. LEE MYERS, M.D., St. Louis, Mo.: Those of us who have worshipped at the shrine of Doctor Mosher know that there are entirely too many cases of esophageal cancer for which nothing can be done. My surgeon friends complain that these cases are referred to them entirely too late. Even at that, I have seen cases where the length of time the patient had been unable to swallow was as short as one week. Doctor Mosher states that frequently an esophagus with an opening 4 mm. in diameter (pipe-stem) will give very few symptoms. In the past the best I have been able to do for these patients has been to perform gastrostomies and administer an occasional dose of radium contained in a capsule. There has been a noted reluctance on the part of surgeons even to attempt surgery of this sort. Many of us stand by and await the grim Reaper, feeling that our help is of poor quality.

In passing, I wish to sound a note of warning to esophagologists to the effect that the slightest injury to the esophagus manifested by signs of perforation, i.e., emphysema, etc., should resolve in a hurried consultation with the thoracic surgeon. Many fatalities following such injury have been prevented by immediate surgical intervention with the recovery of the patient in a manner nothing short of being miraculous.

RICHARD H. SWEET, M.D., Boston: I felt, in coming to Chicago to talk about carcinoma of the esophagus, like one bringing coals to Newcastle, because it was, in part, Doctor Phemister's work with the condition which stimulated me to take it up, and I am grateful to him once again for his kindness in discussing my paper.

I am also grateful to Doctor Mosher for his elucidation of how it happened that I was invited to speak here. I must say, until this moment, that I hadn't quite realized how it came about.

Now, Doctor Phemister has left, but I would like to reiterate his statement that the handling of these mid-esophageal tumors is in a phase of development, and I am certain that what I do today is not what any of us will probably be doing 5 or 10 years hence.

Already it has become apparent, with a small group of cases, that the individual problems vary so much that one must be versatile enough to modify the technical details from case to case. Actually, as a general principle, it would seem to me that in any organ of the body, the more of that organ (if it happens to be involved by cancer) that one can remove, the better. In cases of carcinoma of the thoracic esophagus, I believe that the ideal thing will be to carry the resection from the highest possible point, perhaps 2 to 4 cm. above the arch of the aorta, in order to give a satisfactory cervical esophagostomy downward, to and including the cardiac end of the

stomach, so as to remove the entire lower portion of the esophagus as well.

If one is to do that, then the advantages of the Beck-Jianu type of gastrostomy will be apparent because it makes one independent of utilization of the lower portion of the esophagus for the lower stoma. That can be completely done away with.

Doctor Phemister spoke of dividing the thoracic duct. I have seen the thoracic duct once. I have cut it once, and in that case no harm resulted. In the other cases I wasn't sure whether I saw the duct or not, and apparently didn't injure it.

I am grateful to Doctor Woodward and also to Doctor Myers for stressing the importance of early diagnosis. Obviously those of us who are called upon to do the surgical resections are anxious to get the patients as early as possible. And I have been impressed, as Doctor Myers has, with the fact that even though the history may frequently be short, the stage of the disease is advanced. I presume that Doctor Mosher's teaching about the lumen of the esophagus being so narrow before any obstructive symptoms result is the answer to that problem. In some way, though, we must strive to get the patients earlier.

May I conclude, Doctor Mosher, by saying that I really did not have time to go into the technical matters in any great detail.

UVEITIS WITH SECONDARY GLAUCOMA

F. Bruce Fralick, M.D.
and
James H. Cooper, M.D.
Richard C. Armstrong, M.D.
BY INVITATION
ANN ARBOR, MICHIGAN

Iridocyclitis is so commonly seen by the practicing ophthalmologist that he is usually alert to possible complications. He has learned by experience that early and energetic treatment must be instituted to avoid complications. Some complications most frequently have developed before the patient comes under observation, and occasionally develop in spite of the most expert care. Early recognition of iridocyclitis is imperative and usually must be accomplished with the aid of the slit lamp. It is lamentable, therefore, that so few ophthalmologists have the needed equipment to permit early differentiation and treatment. Complications of iridocyclitis tax the ingenuity of the oculist to the utmost to correct the damage done.

Of the many sequele possible, secondary to primary iridocyclitis, we wish to consider only glaucoma. No mention will be made of postoperative, posttraumatic, or collateral inflammations of the iris and ciliary body. We chose this particular topic for study because we have frequently found ourselves baffled by the destructive processes continuing in spite of all forms of treatment. In certain difficult conditions it is sometimes good to review the results of treatment that has not been too satisfactory, and put them before your colleagues for constructive criticism.

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The most commonly accepted causes of primary iridocyclitis are syphilis, tuberculosis, focal infection, arthritis, gonorrhea, herpes, sarcoid and rheumatic conditions. Many other causes are recognized but do not occur often enough to be of importance in either office or clinic practice. A search of the literature reveals no consistent figures of frequency in the various etiologic factors and will not be considered in this discussion.

The iris is made up of blood vessels and smooth muscle fibers held together by a very loose, spongy stroma. Inflammation of the iris is essentially the same process that occurs in other connective tissue; namely, dilatation of the blood vessels, impairment of the capillary walls, production of a highly viscous albuminous lymph into the tissue spaces and either leukocytic or lymphocytic infiltration. Due to the vascularity of the iris, the distribution of the vessels and the looseness of the connective tissue stroma, distinctive results are seen when inflammation takes place. The increased vascularity of the iris causes a greater amount of exudation than ordinarily seen in connective tissue reaction. The heavy albuminous exudates find their way into the aqueous, increasing its specific gravity and turbidity, as evidenced by the slit lamp. The increase in specific gravity, fibrin and cellular content of the aqueous makes its escape through the filtration angle more difficult and may in turn produce a second-

ary rise in intraocular tension. Since these aqueous changes are the usual findings in acute iridocyclitis, whether intraocular tension is elevated or not, they cannot be the only factors at work. The increased edema of the iris may act in two ways to produce a rise in ocular pressure. First, the periphery of the iris is brought in close contact with the anterior lens capsule, thus paving the way for the formation of posterior synechia. Second, the base of the iris is pushed forward to encroach on the filtration angle, or even obliterate it, by the production of anterior root synechia. These changes may come on rapidly in the acute forms, or more slowly in the subacute or chronic forms of iridocyclitis. A tonometer should be used freely as the only accurate means to early discovery of secondary rise in tension.

The medical treatment of glaucoma secondary to active primary iridocyclitis involves cautious but fearless use of atropine. Only too often we have had patients sent in with acute iridocyclitis and secondary glaucoma using miotics to no avail. Posterior synechiae have been allowed to develop and subsequent management thereby complicated and prolonged. It is our belief that where the rise in intraocular tension developed during the acute phase of iritis, the response to treatment is usually very satisfactory. Homatropine may be prescribed and the patient kept under close observation until therapeutic effects are noted. It is noteworthy, however, that homatropine may have no apparent mydriatic effect on an acutely inflamed iris, especially if there is posterior synechia. In such instances atropine should be used. In contradiction to Gifford's[4] findings we use 1:1000 or 1:100 epinephrine or its substitutes in the form of conjunctival packs in the upper and lower fornices to fortify the action of the homatropine or atropine where needed, and have experienced no further uncontrollable rise in tension. When the tension is depressed by these preliminary procedures atropine may safely be used to maintain dilatation. The usual procedures carried out for uncomplicated iridocyclitis constitutes the further treatment. Where the tension remains elevated in spite of the medical therapy in those eyes showing active uveitis, paracentesis is indicated. It is logical first to attempt reduction of the tension with miotics and to use mydriatics as a secondary measure, if needed, where there is no active iridocyclitis but an elevated tension.

We wish to emphasize the use of typhoid vaccine in iridocyclitis. We feel certain that its use, when followed in two days by paracentesis of the cornea, has materially lessened the duration of the iridocyclitis. Many consider paracentesis a dangerous procedure, but we have not found this to be true.

A. L. Brown[2] has shown that after giving typhoid antigen the blood titre may be as high as 1:480 while that of the aqueous is only 1:2 or 1:4. The secondary aqueous shows a marked increase in antibody titre by paracentesis of the cornea, and the results of foreign protein therapy are greatly enhanced.

At this point, we would like to offer a word of warning in the use of any form of foreign protein, whether given intravenously, our method of choice, or intramuscularly. Most of the articles we reviewed on the use of foreign protein in iridocyclitis mentioned no possible anaphylactic reaction. After giving many hundreds of injections of typhoid vaccine and typhoid H. antigen intravenously without first skin testing the patient for hypersensitivity, we got a near mortality from its use. It is a common misconception among the medical profession that the dosage, manner of giving, or type of foreign protein used play a definite part in the probability of anaphylactic reaction. Intradermal skin testing for anaphylaxis should remove all fear from the use of foreign protein.

The use of hypertonic solutions (50 c.c. of 50 per cent sorbital or 50 per cent dextrose) intravenously are advocated by Bellows.[1] Our experience with its use in secondary glaucoma has been disappointing. The use of 10 to 20 minims of 20 per cent mecholyl as a retrobulbar injection, recommended by Clark[3], has not enhanced the effectiveness of our medical management of glaucoma secondary to primary iridocyclitis.

Failure of medical measures to reduce the intraocular tension below 35 millimeters of mercury (Schiotz) in acutely inflamed eyes indicated that surgical measures should be instituted. Delay in surgical control results in further structural changes in the eye and a higher percentage of unsuccessful results. Failure of medical measures to reduce the intraocular tension below 30 millimeters of mercury (Schiotz) in quiescent iridocyclitis with secondary glaucoma indicates to me that further visual loss will result if surgical intervention is not instituted.

The surgical treatment of iridocyclitic secondary glaucoma resolves mainly into the use of 2 procedures: paracentesis and iridectomy. We believe that eyes showing iridocyclitic glaucoma should not be subjected to surgical procedures unless definitely indicated. When indicated we have resorted to paracentesis in eyes in which the condition is acute or subacute and in which it cannot be controlled by medical measures. It would be difficult to conceive any lasting benefit from paracentesis where organization has occurred in the filtration angle. Where foreign protein has been used previously, evacuation of the anterior chamber washes out the cellular and fibrinous aqueous, clears the filtration angle and increases the antibody titre. It is best done subconjunctivally in the corneal periphery. The aqueous should be allowed to escape slowly. The congested iris under high tension is very prone to show interstitial hemorrhage or hemorrhage into the anterior chamber if the globe is decompressed so rapidly that the blood vessels have no chance to compensate for the change in intraocular tension. Should the tension rise again the anterior chamber may be re-opened by passing a spatula between the lips of the paracentesis wound. This is repeated, within reason, until the iritis is under control, lessening the edema and infiltration of the iris and the cellular and fibrinous exudate, all of which play a part in promoting rise in tension.

An iridectomy is the most logical procedure in those eyes where the residues of inflammation do not resolve or where medical management and paracentesis fail. Iridotasis and iridencleisis are not well adapted to this problem because the incarceration, under the conjunctiva, of an iris which is acutely or chronically inflamed, vascularized or degenerated, does not result in a satisfactory filtrating apparatus. Similar poor results are to be expected in manipulating an inflamed ciliary body in cyclodialysis. Iridectomy should be the primary procedure in glaucoma that has been present for some time and where the inflammation has, or nearly has, subsided.

Corneoscleral trephining offers the best results of any of the filtering operations but should only be applied after the inflammation has subsided. It has been shown that the exudates coalesce the ciliary processes into a plug which may close the trephine opening with consequent failure of the operation. Some consider the presence of anterior root synechia in the presence of secondary glaucoma, as found by gonioscopy, to be indication for either the LaGrange sclerotomy or corneoscleral trephine.

Transfixion is indicated where complete posterior synechia with consequent iris bombé is present. It may be preferable here at times to perform a complete or basal iridectomy through a subconjunctival scratch incision at the limbus

either because of the possibility of trauma to the lens in transfixion or because of a personal dislike for the usual transcorneal incision.

Statistics on the results of medical and surgical treatment in iritic glaucoma are found with difficulty. Larsen[5] studied a series of 50 iritic glaucoma eyes and found the results were perfectly satisfactory in only 2 out of 19 iridectomies, and the results after trephining were even poorer than with iridectomy. It must be pointed out, however, in some instances that the trephines were done on eyes which had failed to respond to iridectomy.

A survey of 1,282 consecutive cases of iridocyclitis revealed that 261, or 22 per cent, had secondary glaucoma or developed the condition while under observation. This is undoubtedly a conservative estimate, as 62 were sent in for diagnosis only, returned home for subsequent treatment and may in some instances have developed glaucoma after passing from our observation.

Primary Iridocyclitis	1282	
With Secondary Glaucoma	261	(22%)
Glaucomatous Eyes Treated	199	
Glaucomatous Eyes Diagnosed Only	62	

TABLE 1.—Table of eyes showing primary iridocyclitis used in study. The glaucoma group is made up of 257 individuals, 4 having bilateral disease. In the statistical study made each eye is considered as a separate patient. Sixty-two of the patients with glaucomatous eyes were sent in for diagnosis only and were not treated by us.

The ratio of female to male in the glaucoma group was approximately 2 to one. The highest age incidence fell in the sixth decade, the mean age being 53 years. The youngest patient was 20 and the oldest 76.

In no patient was the etiology of the iridocyclitis in the glaucoma group definitely proven. However, a definite focus of infection was found in over one-half (100 to be exact) of the patients who came in for treatment. The routine search for focal infection included examinations by otolaryngogists, oral surgeons, and gynecologists or urologists, and the Kahn test, together with tuberculin tests and chest x-rays, were indicated.

Of the 199 eyes with secondary glaucoma, 69 were treated medically as the primary procedure. This type of treatment was carried out over a sufficient period of time to enable evaluation of the results.

Treatment used	*T. Normal*	*T. Elevated*	*Total*
Mydriatics	17	29	46
Miotics	6	15	21
Miotics then Mydriatics		2	2
Total	23 (33%)	46 (66%)	69

TABLE 2.—Medical treatment of primary iridocyclitis with secondary glaucoma. Two actively inflamed eyes were unsuccessfully treated, first by miotics, and then by atropine. The tension of one of these eyes was subsequently normalized by iridectomy, while the second eye was blind and, therefore, enucleated. It probably should have been enucleated as the primary procedure.

The remaining 67 eyes treated medically were given either mydriatics or miotics, and in no other instances were both used. Either homatropine, when effective, or atropine was used in 46 eyes showing acute or moderately active inflammation and in only 17 instances was the tension reduced to below 30 millimeters of mercury (Schiotz). Miotics were tried in 21 eyes showing more or less quiescent iridocyclitis, and the tension was reduced to normal in only 6 eyes. The question naturally arises as to why a mydriatic was not used if miotics did not succeed in reducing the tension. It is quite likely that medical therapy of iritic glaucoma would possibly have made a better showing if a more diligent attempt to reduce the tension had been carried out this way. Our first problem in iridocyclitic glaucoma is to dilate the pupil, thus combating the uveitis causing the glaucoma. During the active stage of uveitis, posterior synechia should be prevented and where already

present should be ruptured when possible by mydriatics or packs of adrenalin applied to the conjunctival cul-de-sac. One may be justified in using miotics primarily in quiescent iridocyclitic glaucoma, but one cannot continue miotics long in an acutely inflamed eye without posterior synechia developing and further complicating the course of the disease.

In 58 eyes, all showing some degree of activity of iridocyclitis, paracentesis was performed.

Treatment used	*T. Normal*	*T. Elevated*	*Total*
Paracentesis	25 (43%)	33 (57%)	58

TABLE 3 — Surgical treatment of primary iridocyclitis with secondary glaucoma. It was surprising to find that in only 25 eyes normal tension was obtained without more radical surgery.

In several eyes (with a maximum of 9) paracentesis was often repeated. It was repeated more than once in only 6 of the 25 eyes where it sufficed as the only operation necessary. In these 6 eyes, 2 required 2 paracenteses, 3 required 3 paracenteses, and one required 4 paracenteses.

Frequency of Paracentesis	1	2	3	4	5	6	7	8	9	Total
T. Normal	19	2	3	1						25
T. Elevated	7	15	3	4		3			1	33
Total	26	17	6	5		3			1	58

TABLE 4 — Surgical treatment of primary iridocyclitis with secondary glaucoma.

This might suggest that if paracentesis is to be successful, one or 2 paracenteses at the most is sufficient, and that some other procedure might better be utilized after 2, rather than to continue to subject the eye to repeated trauma with little hope of success. However, in acute uveitis it must be remembered that paracentesis may remain the least radical means of controlling the tension until the inflammation has subsided sufficiently to permit some other means of control.

Iridectomy was used in 60 eyes, controlling the tension in 43 and failing in 17. In 28 this was the initial procedure and in the remaining 32, 13 were preceded by medical therapy, and 19 by paracentesis.

Treatment used	*T. Normal*	*T. Elevated*	*Total*
Iridectomy			
Primary	20	8	28
After Medical	10	3	13
After Paracentesis	13	6	19
Total	43 (71%)	17 (28%)	60

TABLE 5 — Surgical treatment of primary iridocyclitis with secondary glaucoma.

The first group consisted of 20 successes and 8 failures with iridectomy as the initial procedure. We have considered a result successful if the tension remained within normal limits during the period it could be followed. Of these 28 eyes the iritis was inactive in 23 and either active or questionably so in 5. It was interesting to note that the 8 failures were equally divided between the inactive and the actively inflamed eyes.

The second group consisted of 32 eyes, where iridectomy was either preceded by medical therapy or paracentesis. In 10 out of 13 eyes unsuccessfully treated by medical therapy the tension was normalized by iridectomy. In this group 8 eyes were not considered actively inflamed, with 5 actively inflamed. One failure occurred in a quiet eye and 2 occurred in actively inflamed eyes.

The tension was normalized in 13 out of 19 eyes in which iridectomy was performed after paracentesis had failed. All 6 failures showed some degree of activity of the iridocyclitis, there being only 3 successes in the inflamed group. From these findings we might conclude that the chances for reduction in tension by iridectomy are greater if the ocular inflammation is decreased as much as possible before iridectomy.

In summarizing all 60 iridectomies, whether done initially or finally, 43, or 71 per cent, were successful and 17, or 28 per cent, were failures. On the whole, very gratifying results in glaucoma, secondary to primary iridocyclitis, are given by iridectomy when indicated through the failure of medical therapy or the conservative operation of paracentesis.

A single trephine operation was done in 15 eyes, 9 of which resulted in reducing the intraocular tension to normal.

Treatment used	*T. Normal*	*T. Elevated*	*Total*
Trephine			
Primary	4	4	8
After Paracentesis	1	2	3
After Iridectomy	4		4
Total	9 (60%)	6 (40%)	15

TABLE 6 — Surgical treatment of primary iridocyclitis with secondary glaucoma. All eyes trephined showed no activity of the iridocyclitis. In 8 instances trephine was used as the initial operation and was successful in half the eyes. In 5 of the 9 instances where trephine apparently controlled the tension, it had been preceded by either single or multiple operations. Four of the trephines were preceded by iridectomy, one by paracentesis.

In a miscellaneous group of operations, done more or less as a last resort in eyes having a very poor prognosis or after other operations had failed, we were much surprised to find that collectively they resulted in 23 successes and 14 failures.

Treatment used	*T. Normal*	*T. Elevated*	*Total*
Cyclodialysis			
Primary	4	3	7
After Paracentesis		2	2
After Iridectomy	5		5
After Trephine	1	3	4
Total	10 (56%)	8 (44%)	18
Transfixion	2 (67%)	1 (33%)	3
Combined			
Cataract Ext.	9 (75%)	3 (25%)	12
Iridencleisis	2 (50%)	2 (50%)	4
Total	23 (62%)	14 (38%)	37

TABLE 7 — Surgical treatment of primary iridocyclitis with secondary glaucoma. In this group there were 18 quiescent eyes operated by cyclodialysis and in 10 the tension was reduced to normal during the period of observation. Seven of these operations were done as the primary procedure and 4 resulted in normal tension. Six of the remaining 11 eyes responded with normal tension after other types of operations had failed (4 after trephine, 5 after iridectomy and 2 after paracentesis). Two transfixions of the iris for iris bombe were successful and one failed — all being the primary operation used. Normal tension resulted in 9 out of 12 primary combined extractions of complicated cataract with iritic glaucoma. Iridencleisis as the primary procedure in 2 eyes was successful and a failure in 2 following trephine.

Sixty-seven eyes were enucleated in this series. Thirty-five were considered hopelessly lost and enucleated as the primary procedure. Eight additional eyes were considered hopelessly lost but at the request of the patient, medical therapy was tried but without success for a short period of time before enucleation. One or more surgical procedures were carried out on the 24 remaining eyes before they were given up as hopeless.

Treatment used	*Eyes Lost*
Enucleation	
Primary	35
After Medical	8
After Other Surgery	24
Total	67 (25%)

TABLE 8 — Eyes having primary iridocyclitis with secondary glaucoma requiring enucleation.

CONCLUSIONS

1. Glaucoma secondary to primary iridocyclitis occurred in 22 per cent of 1,282 eyes examined.

Treatment used	*T. Normal*	*T. Elevated*	*Total*
Medical	21 (30%)	48 (70%)	69
Paracentesis	25 (43%)	33 (57%)	58
Iridectomy	43 (71%)	17 (29%)	60
Trephine	9 (60%)	6 (40%)	15
Cyclodialysis	10 (56%)	8 (44%)	18
Transfixion	2 (67%)	1 (33%)	3
Combined			
Cataract Ext.	9 (75%)	3 (25%)	12
Iridencleisis	2 (50%)	2 (50%)	4
Enucleation			67
			or (25%)

TABLE 9 — Summary of medical and surgical treatment of primary iridocyclitis with secondary glaucoma.

2. Medical therapy consisting either of mydriatics or miotics, or a combination of both, in addition to heat, salicylates, foreign protein, and re-

moval of focal or general infection resulted in normalization of the intraocular tension in only 30 per cent of the eyes.

3. Paracentesis resulted in normal tension in 43 per cent of the eyes when used in active iridocyclitis accompanied by glaucoma. It should, we believe, be the primary surgical procedure used in this group.
4. If paracentesis is to be successful one or 2 paracenteses at the most is usually sufficient and after failure of paracentesis on 2 occasions, some other procedure might better be utilized unless the eye shows active inflammatory changes contraindicating other surgery.
5. Of all operative procedures, iridectomy gave the highest percentage (71 per cent) of controlled intraocular tension in iritic glaucoma. If the ocular inflammation is first decreased to a minimum favorable reduction in tension by iridectomy is greater.
6. Corneoscleral trephine was successful in reducing elevated intraocular tension in 60 per cent after the iridocyclitis was quiescent. This operation is not indicated in eyes showing activity of uveitis.
7. Cyclodialysis, iridencleisis, transfixion of the iris and combined extraction of complicated cataract in quiescent iridocyclitis with secondary glaucoma resulted collectively in reducing the tension to normal in 62 per cent of the eyes operated.
8. Sixty-seven (25 per cent) of the eyes showing glaucoma after the development of primary iridocyclitis were primarily or finally enucleated.

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DISCUSSION

PETER KRONFELD, M.D., Chicago, Illinois: One of the many annoying and exasperating features of iridocyclitic secondary glaucoma is the extreme variability and irregularity of the disease. If in a case of iridocyclitis the intraocular pressure becomes elevated, it is difficult to predict whether this is going to be just a brief episode or whether this will prove a permanent secondary glaucoma, resistant to all forms of medical and surgical therapy which are at our disposal. Even the gonioscopic findings do not always enable us to tell with certainty whether the anatomy of the chamber angle has been altered to such an extent that the function of the normal channels of outflow is permanently or irreparably impaired. Thus in iridocyclitic secondary glaucoma we have to act from day to day, or rather from week to week, since, with the only exception of seclusion of the pupillae with iris bombé, no situation in iridocyclitic glaucoma calls for immediate surgical action. The well-known tolerance of the human eye to such elevations of intraocular pressure and the possibility of nature coming to our help justify the conservative attitude which is so well expressed by the term "sitting tight."

I have seen several cases of more or less quiescent iridocyclitis, in which an elevation of intraocular pressure, despite intensive medical treatment, persisted for up to 8 months and then subsided with hardly any permanent loss of visual function. Our experiences at the Illinois Eye and Ear Infirmary are in close agreement with those of Doctor Fralick. In glaucoma associated with quiescent iridocyclitis, cyclodialysis has in our hands scored relatively more successes than a trephine operation. The difference, however, is not great enough to permit the statement that cyclodialysis is the most suitable operation for this type of glaucoma, nor do we have any data, gonioscopic or clinical, which enables us to tell beforehand which of the

two operations, trephine or cyclodialysis, is more likely to be successful in a given case.

In a case of very extensive peripheral anterior synechia, especially above, in the upper portion of the angle, I would prefer the cyclodialysis to the trephine.

In each of Doctor Fralick's series of five cases cyclodialysis after iridectomy was successful. I wonder if Doctor Fralick attaches any significance to this observation. Was the cyclodialysis made in the area of the coloboma? If so, the success of these operations might have been due to the effect of the iridectomy combined with the cyclodialysis, the procedure advocated by Czermak, Wootton, Wheeler, and recently by Goldmann and Bangerter. In Doctor Fralick's series, the iridectomy alone makes an unusually good showing. I wonder if very extensive posterior synechiae were characteristic of the cases in which iridectomy alone controlled the glaucoma.

I would like to take issue with only one thing in Doctor Fralick's paper, namely, the use of the word "only" in the following sentence: "Paracentesis was performed in 58 eyes showing some degree of active uveitis. I was surprised to find that in only 25 eyes normal tension was obtained without more radical surgery." Does such a percentage of successes in a procedure as simple and harmless as paracentesis justify the use of the word "only" if a much more extensive procedure like a trephine was successful in 60 per cent?

Doctor Fralick's experience that paracentesis repeated several times at short intervals does not tend to be any more effective than just one or two paracenteses agrees entirely with our experience. It, therefore, seems possible that paracentesis acts largely by breaking a vicious circle. Our experiences with the cyclo-diathermy of Vogt are sufficient to recommend its trial if other surgical procedures have failed.

The period of postoperative observation in our cases has not been long enough to warrant my presenting any results.

In my opinion, Doctor Fralick and his associates have made a contribution of great practical value in a field where every little bit of guidance and advice is very keenly appreciated.

F. Bruce Fralick, M.D., Ann Arbor, Michigan: I thank Doctor Kronfeld for his discussion of this paper. He asked the question whether the cyclodialysis opening was made in the region of the coloboma of the previous iridectomy. I cannot answer that question definitely, but it is my impression that the cyclodialysis opening was not uniformly made in the region of the coloboma but more commonly in the outer lower quadrant.

He also brings up the question as to whether posterior synechiae were not common in the cases in which iridectomy was performed. It is certainly true that the majority of the patients requiring iridectomy, or in whom irridectomy was done, did show a high percentage of relatively advanced posterior synechia and that may logically be an explanation for the high percentage of results in that group. However, this group did not show the characteristics of iris bombé so that you would get the end result of a transfixion.

We also have been very favorably impressed by the results of cyclo-diathermy cauterization in the quiescent form of iridocyclitic secondary glaucoma. I have, however, not used it on eyes which have very good vision. Most of the patients operated upon by this procedure have been those not responding to the other forms of treatment and thus cyclo-diathermy cautery was selected as a last resort. We have been very favorably impressed with the results in this particular group and may later gain sufficient courage to extend the use of the operation to the less hopeless groups.

WHY A GLAUCOMA CLINIC

HARRY S. GRADLE, M.D.
and
H. ISABELLE MCGARRY, M.D.
BY INVITATION
CHICAGO, ILLINOIS

IN 1937 a survey of the cases of glaucoma seen at the Illinois Eye and Ear Infirmary during the preceding four years revealed the most disquieting fact that only 4 per cent remained under observation for 2 or more years. The visual fate of the other 96 per cent was unknown. So with money obtained from the Otho Sprague Foundation and from the Illinois Society for the Prevention of Blindness, a Glaucoma Clinic was opened at the Infirmary in February, 1939. The Glaucoma Clinic is a supplement to the regular ophthalmological services of the Infirmary and serves to keep all cases of glaucoma, under medical and social observation, under the direction of the regular Attending Ophthalmologists of the Infirmary. The Glaucoma Clinic neither treats nor operates.

As many patients have unilateral glaucoma and others have only one eye, it is necessary in this communication to refer to individual eyes rather than individual patients.

This report deals with the first 495 eyes observed in the Glaucoma Clinic, divided as follows:

Primary Glaucoma, the group considered in the following statistical analysis....	283 eyes
Secondary Glaucoma, not considered in this report.....	45 eyes

Not considered in this report because of:

1. Discharged to private physician	34	eyes
2. Blind eyes	54	
3. Health too poor to return to clinic	28	
4. Changed to another clinic	14	
5. Died	 8	
6. Moved out of State.....	4	
7. Moved and could not be found	 4	
8. Refused to return	21	167 eyes
Total	495	

So items 7 and 8 show that only 25 eyes out of a total of 495 (5 per cent) became statistically lost. This is an exact reversal of figures that pertained before the Glaucoma Clinic was instituted.

The 283 eyes with primary glaucoma considered in this report have all been under observation for a minimum of 2 years. It is realized full well that this period is too short to permit of absolute conclusions, but this is somewhat in the nature of a preliminary report compiled to a great extent for our own guidance. Furthermore, it is obviously impossible to compile accurate statistics upon so variable a condition as glaucoma. For example, an eye with chronic glaucoma may suddenly flare up with an acute congestive attack; an acute case with only light perception upon admission may be brought under control with resultant vision of 20/20; a chronic case may have reduced vision upon admission due to cataract which in the course of time becomes mature so that the lens

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can be extracted with resultant vision of 20/40 or better; and so on. Such incidents multiply during the course of 2 or 3 years' observation to such an extent that only generalities can be dealt with statistically.

Of these 283 eyes with primary glaucoma, 13.8 per cent were of the acute and 86.2 per cent of the chronic type.

Treatment was dictated by the judgment of the Attending Ophthalmologist and hence was not uniform. In 40 per cent of the cases, the initial course of treatment was with miotics alone. Twenty per cent were operated upon immediately and required no further treatment. The remaining 40 per cent were operated upon, but required further use of miotics. Naturally, these figures changed as the demands of the individual case indicated, consequently the results to be reported must be considered as the result of combined treatment and not of miotics alone or of surgery alone.

Of course, the ideal way to determine the results of controlled glaucoma therapy would be to measure the visual efficiency, using the formula of the Committee on Compensation for Ocular Injuries of the American Medical Association. That formula is based upon weighted values of central visual acuity for distance and for near and upon value of the visual field. But this was not possible or practicable in these cases. So central visual acuity for distance, measured on the Snellen Chart, was used as the basis of compilation. The visual field values are considered separately and will be discussed later.

VISUAL ACUITY

Table 1 shows the visual fate of glaucomatous eyes with visual acuity of 20/20 to 20/50 inclusive at time of admission to the Clinic.

TABLE I

	ON ADMISSION	AFTER 12 MONTHS	AFTER 18 MONTHS	AFTER 24 MONTHS
20/20-20/50 inc.	148	124	116	117 (79%)
20/50-20/70		8	10	9
20/70-20/200		11	12	10
20/200 or less		5	9	12(8.6%)

Seventy-nine per cent of the eyes in this group retained the original acuity of vision during the 2-year period of observation; 8.6 per cent lost visual acuity to a point of industrial blindness; and 12.4 per cent sustained some loss. Roughly summarized, one may say that 80 per cent of all eyes, with primary glaucoma with good vision (20/50 or better) at the time treatment is instituted, will retain that vision for at least 2 years, and that 91.4 per cent will retain useful vision (better than 20/200).

Table II deals with glaucomatous eyes in which the vision was less than 20/50, but better than 20/200 at the time of admission. In other words, these were eyes in which sufficient damage had been done to cause a definite visual handicap, but still were not industrially blind.

TABLE II

	ON ADMISSION	AFTER 12 MONTHS	AFTER 18 MONTHS	AFTER 24 MONTHS
From 20/50 up to 20/200	42	23	25	26 (61.9%)
20/200 or less		19	17	16 (38.1%)

As can be seen, 61.9 per cent retained after 2 years approximately the same

degree of vision as at the time of admission, whereas 38.1 per cent became industrially blind. Roughly summarized, one may say that of the eyes more severely damaged by the disease before treatment was instituted, only 60 per cent retained useful vision as contrasted to the more than 90 per cent of Table 1. By the same token, 40 per cent became industrially blind while of the better eyes of Table 1, only 8 per cent suffered that fate. These figures reveal most graphically that the salvation of vision in glaucoma depends upon the institution of treatment before severe visual damage has occurred; that is to say, the early discovery of the disease.

In Table III, only eyes that were industrially blind from the onset of treatment are considered.

TABLE III

	ON ADMISSION	AFTER 12 MONTHS	AFTER 18 MONTHS	AFTER 24 MONTHS
20/200 or less	66	59	57	56 (80.4%)
No L. P.		1	2	4 (6.0%)

The apparent discrepancy in figures is explained by the fact that 6 of the eyes in this group were enucleated. But the latter figure is of minor importance as contrasted to the fact that 80.4 per cent of the eyes in this class retained visual acuity equal to that on admission.

Twenty-seven eyes had no light perception upon admission. Of that number 13 required enucleation for one reason or another.

Thus the 283 eyes with primary glaucoma can be divided into 4 main groups, based upon the central visual acuity at the time treatment was first started, as follows:

	No.	*Percentage*
Vision of 20/20 to 20/50 inclusive	148	52.3%
Vision of less than 20/50 but better than 20/200	42	14.9%
Vision of 20/200 or less	66	23.3%
No light perception	27	9.5%

Of the 190 eyes with admission vision of 20/20 up to 20/200, only 28, or 14.7 per cent (roughly 15 per cent), suffered loss of central visual acuity to less than 20/200, or industrial blindness.

It is difficult to reconcile the figures of these different tables and absolutely impossible to plot them into any curve. But taking into account the comparatively small number and the fact that the eyes were observed for only 2 years, it is still possible to obtain an estimation of what will be the fate of an average glaucomatous eye based upon vision at the time treatment was instituted.

It was not possible to establish a basis for the comparison of central fields. Evaluation of the peripheral visual fields was an even more difficult matter. The amount of loss was estimated somewhat roughly upon the amount of contraction in the eight principal meridians and expressed in terms of percentage. Full fields to about 25 per cent loss were considered normal whereas from 75 per cent to 100 per cent loss was evaluated as practically complete loss. Table IV shows the number of cases that sustained partial or complete loss of visual peripheral fields during the 2 years covered by the observation.

TABLE IV

% Loss	ON ADMISSION	AFTER 12 MONTHS	AFTER 18 MONTHS	AFTER 24 MONTHS
No Loss to 25%	107	80	80	72 (67.3%)
25-50%	33	42	40	43
50-75%	35	43	39	37
75-100%	107	114	119	124 (14.1%)

Of the 107 eyes with normal fields at the time of admission, 72, or 67.3 per cent, suffered no loss during the 2 years. One hundred and seven had already lost from 75 per cent to 100 per cent of the fields before admission and that number was increased in 2 years' time to 124; in other words, only 17 (14.1 per cent) additional eyes suffered complete loss of fields. Eyes with a partial loss of field at the time of admission showed improvement in many cases and in only 18.6 per cent was there further loss. To summarize roughly, of the glaucomatous eyes with normal fields when treatment was instituted, approximately 70 per cent retained those fields whereas in only 14 per cent was the visual field completely lost.

It is impossible to compare these conclusions with other series of cases for as far as a search of the literature reveals, a similar analysis has never been made. Numerous compilations of end results from the standpoint of the control of intraocular pressure subsequent to this, that, or the other operation have been published, but a generalization as to the visual fate of the glaucomatous eye, based upon statistical data, has never appeared. Five years from now, and again 5 years after that, further analysis of these same eyes must be made, and in that study must be included end results according to the various types of glaucoma, according to the specific treatment employed, and according to the initial age of the patient.

CONCLUSIONS

1. The Glaucoma Clinic, operated as a supplement to the regular Ophthalmological Services of the Illinois Eye and Ear Infirmary, has proven to be of value in the following ways:
   A) Ninety-five per cent of the glaucomatous eyes are kept under necessary observation for 2 or more years, whereas previously 96 per cent of them disappeared within 2 years.
   B) It has served as a Training Center in the diagnosis and clinical study of hypertensive eyes for nurses, social workers and physicians.
   C) It has provided material for extensive investigative work in various aspects of glaucoma.
2. Analysis of the first 283 eyes with primary glaucoma of both acute and chronic types, observed over 2 years, shows that:
   A) The better the visual acuity and the visual fields at the time of discovery of the disease, the better is the chance of retention of useful vision by continued observation and treatment.
   B) Of all the eyes with useful vision only 14.7 per cent became industrially blind within 2 years.
   C) The loss of vision in the peripheral field precedes the loss of central visual acuity from glaucoma.
3. These figures emphasize that the prevention of blindness from glaucoma is dependent upon the early recognition of the disease and that the efforts of ophthalmologists must be directed with fuller force toward the education of lay and medical personnel with that aim in view.

DISCUSSION

MARK J. SCHOENBERG, M.D., New York: Doctors Gradle and McGarry have read a most interesting paper. I do not see where anything more could be added to what they had to say.

Last year, the chairman of the Committee on Glaucoma for Greater New York, sponsored by the National Society for the Prevention of Blindness, read before the American Ophthalmological Society a report on a survey of glaucoma records of three New York eye clinics and one eye clinic of Boston. The

greater part of this paper is a document illustrating how unsatisfactory and chaotic glaucoma work can be, if it is conducted along old, traditional lines, which have remained almost unchanged for the last 100 years. On the other hand, the study of the Boston clinic demonstrates that it is not impossible to reform the old ways and that better results are obtainable by employing more up-to-date methods, just as Doctors Gradle and McGarry have assured us today in their paper. I wish to add that conditions are improving fast at New York City eye clinics.

As an introduction to discussion of the paper, allow me to relate some of my own experiences.

In October, 1935, a special clinic for glaucoma patients was opened at the Knapp Memorial Eye Hospital of New York. The staff of the clinic consisted of a director, an assistant and a medical diagnostician.

This group started out with the idea of studying their patients as thoroughly as the means at their disposal would permit. Perimetry was recorded every 4 to 6 weeks; the patient's complaints, both about the physical and mental condition, were entered on the record no matter how trifling they appeared.

In addition, each member of the staff was charged to study and report on a certain phase of glaucoma. Gradually the work branched out in every possible direction. A social worker and a nurse were added to the staff, and patients were taught how to apply medication to their eyes and the elementary rules of personal hygiene and nutrition. The meaning of glaucoma was explained and the importance of cooperation stressed. Printed leaflets containing the necessary information worded in very plain language were distributed among the patients and members of their families. The clientele of the clinic grew gradually. Thus by organizing a glaucoma clinic, coping with the various practical problems and handling patients in a manner entirely different from the way in which they are being handled in regular clinics and even in private practice, we learned some things one does not find in books or in journals. From this experience the following conclusions were reached:

1. The glaucoma clinic gives an infinitely better service to patients than the average eye clinic.
2. It offers the attending staff — medical and non-medical — the rare opportunity of studying and treating a large number of the same kind of patients within a short time, thereby shortening the span of time necessary to acquire a sufficient amount of knowledge about glaucoma.
3. It serves as a center of education for patients and other lay people who are in need of being informed about prevention of blindness from glaucoma.
4. It could serve as a center of education for medical practitioners and others who are the first ones to come in contact with cases of early glaucoma and on whom we depend for getting the early cases.
5. It could become a center for research, especially clinical research, of the various phases of the glaucoma problem.

In other words: (1) better service to patients; (2) concentrated opportunity of studying glaucoma patients; (3) educational role; (4) center for research.

Let us consider for a few moments the objections to a glaucoma clinic. I believe that very few ophthalmologists will commit the imprudence of denying the importance of organizing special glaucoma clinics. No one could defend successfully the traditional method of handling glaucoma patients, a tradition which has been in vogue for the past century. On the other hand many ophthalmologists doubt that the organization of special glaucoma clinics is a practical possibility. Some of their arguments are:

1. The cost of running such a clinic.
2. The lack of medical and non-medical help.
3. The lack of space.
4. The opposition of the medical staff of the general eye clinic which does not wish to be deprived of the experience of seeing this type of patient, while a small group of ophthalmologists of the glaucoma clinic "monopolizes" the entire clinical and surgical material of glaucoma patients.

The number of those who have condemned innovations in medicine has always been in the majority, and apparently valid objections have always been found even against the most pressing reforms.

The shortage of well-trained professional perimetrists is indeed a serious problem; but even this can be remedied. Lately we have started in New York a course for teaching volunteers the method of perimetry. We hope to have half a dozen perimetrists fairly well trained in about 3 months. They will then be turned over to a few eye clinics, where perimetry is done only casually, to work and to continue their training under the supervision of the attending ophthalmologists.

A small room 10 ft x 10 ft. is sufficient to be fitted out with the necessary implements to serve as a perimetry room. Considering the benefit derived from perimetry, the salary of a professional perimetrist is the best investment an eye institution can make. If there is money for a laboratory, for x-ray, for orthoptic training and aniseikonia departments, there must be money for a perimetry department.

As far as the accusation is concerned that a separate clinic "monopolizes" the glaucoma material, it is based on misinformation and misunderstanding. The staff of the glaucoma clinic is to be on a rotating basis. The members are appointed for 3, 6 and 9 months on a staggered basis so that there are always eye men in charge, who have familiarized themselves with the set-up of the clinic, to teach the newcomers from the general eye clinic the routine of the work and continue the tradition of thoroughness established by the director.

Time does not permit discussion of the subject in greater detail. Allow me to call your attention to the fact that men who have had actual experience with glaucoma clinics should have priority in being heard first. The opinions of our colleagues from the Illinois Eye and Ear Infirmary, Massachusetts Eye and Ear Infirmary, Brooklyn Eye and Ear Hospital, Long Island College Hospital, etc., and my own conclusions should weigh in the balance against those who do not approve of special glaucoma clinics on purely theoretical grounds.

Dr. John Evans wrote to me a few days ago as follows: "I can definitely state that there is absolute agreement at the Long Island College Hospital that the patients are getting far better care in the glaucoma clinic." Dr. Henry Mitchell Smith wrote: "From my experience as director of the special glaucoma clinic at Long Island College Hospital I am firmly convinced that a special clinic is the only way to handle glaucoma cases in a busy eye clinic."

I do not doubt that we all agree that *the real difficulty in running a successful glaucoma clinic is to find a way to discover glaucoma cases in the early stages.* To do this we have to train the public, the general practitioners and optometrists. We have to encourage research work. We should not be satisfied to prevent just blindness from glaucoma. Even 20/100 vision is too low a goal. *We want to save for our patients a normal or very nearly normal acuity and field of vision.*

This discussion should not be concluded without pointing out one weakness in the otherwise excellent paper of the essayists. The weakness is the fact that the authors say: *The Glaucoma Clinic at the Illinois Eye and Ear Infirmary Neither Treats Nor Operates.* In other words, the clinic concentrates its entire attention and limits its activity exclusively to diagnosis. I believe that it is much better and definitely to the advantage of the patients and of the personnel of the glaucoma clinic that the complete work (diagnostic and therapeutic) should be carried out at the glaucoma clinic only. Divided interest and responsibility do not lead to complete efficiency. Perhaps the fact that members of the staff of general eye clinics may fear that they are being deprived of glaucoma surgery has dictated the policy of the essayists of splitting the work between the 2 services. However, this fear is only due to a misunderstanding since, as I have mentioned, one of the main purposes of the special glaucoma clinic is to give an opportunity to every man from the general eye clinic to become proficient in every phase of the glaucoma problem by devoting his time at least once, for a number of months exclusively, to the work in the glaucoma clinic. And this includes — of course — doing also glaucoma surgery.

In Memoriam

JOSEPH C. BECK, M.D.
1870-1942

JOSEPH C. BECK, M.D.
1870-1942

With the death of Joseph C. Beck, on October 20, 1942, American otolaryngology lost one of its pioneers as well as one of its strongest characters.

He was born in Bohemia, September 26, 1870, and the story of his life rivals that of fiction. His career personifies the opportunities of America that are open to those who do truly and earnestly seek them. His childhood was not carefree — his mother died when he was but two years old and his father when he was nine. Perhaps it was the memories of his youth that made him kind and sympathetic and so very human. The story of his childhood, of his youth in America, of his determination to study medicine and of his medical career are well told in his book Fifty Years in Medicine.

After graduating in 1895 from the University of Illinois, he became associated with his brother, Carl, in the practice of medicine. Three years later he decided to specialize, and it is interesting to note that in doing so he decided first to learn about pathology. He went to Prague where he devoted a year to the study of pathology and two years more in preparing for the specialty of otolaryngology. After returning to Chicago his career was one of steady progress. He became associated with his brothers, Emil and Carl, following which the North Chicago Hospital became the Mecca of all visiting otolaryngologists. His interest in pathology continued, and, as a charter member of the American Board of Otolaryngology, he was instrumental in making graduate training in otolaryngology "pathology conscious."

He was a pioneer, too, in former borderline specialties such as plastic surgery, endoscopy and treatment of tumors of the head and neck.

He was a teacher. During his entire medical career he taught. He was associate professor in Otolaryngology from 1901-1932 at the University of Illinois College of Medicine; professor of Otolaryngology from 1902-1914 at the Chicago Eye, Ear, Nose and Throat Hospital, and he gave many instructional courses at annual sessions of the American Academy of Ophthalmology and Otolaryngology. At every opportunity he gave to his students freely of his experiences.

He encouraged reading of the literature. His reading clubs (there were five) met once a month, and Doctor Beck never missed a meeting. There the literature of all languages was abstracted and reviewed.

During World War I Doctor Beck was in charge of the hospital sponsored by the American Red Cross for the Czechoslovakian Government. He served until after the Armistice. He was awarded the Serbian Cross and the Czechoslovakian Revolutionary Medal. Doctor Beck was discharged with the commission of Lieutenant-Colonel in the United States Medical Reserve Corps. After the War his interest in military affairs continued. He became a member and later a commander of the Medical Post of the American Legion of Chicago.

Doctor Beck was the author of eighty or more medical papers, and he perfected many scientific instruments.

He was a member of all, and a past president of most, of the American otolaryngological societies. In 1931 he was chosen Honor Guest of the American Academy of Ophthalmology and Otolaryngology, and he was made a member of the Honor Society of the Academy.

Doctor Beck (to most of his friends and students, "Doctor Joe") loved life and youth. He enjoyed a good story. The movies, music, art, sports and at one time "His Farm" were his hobbies. His influence was widespread. His intense interest, enthusiasm and kindness were genuine. If you were a friend of his you knew it; if there was any doubt about it, you likewise knew it. He was fearless and intellectually honest.

As an expert diagnostician, for ten months he calmly watched his own decline toward death. His passing leaves a great void in our ranks. "Doctor Joe" lived his own obituary:

"To those who knew thee not, no words can paint;
And those who knew thee, know all words are faint."

LeRoy A. Schall

In Memoriam

ALLEN GREENWOOD, M.D.
1866-1942

ALLEN GREENWOOD, M.D.

1866-1942

Allen Greenwood was born in Chelsea, Massachusetts, March 1, 1866. He received his M.D. degree from Harvard Medical School in 1889; was house officer in the Boston City Hospital 1888-1890; instructor in Waltham Training School 1890-1901; bacteriologist Waltham Board of Health 1894-1898; instructor in ophthalmology Harvard Medical School 1904-1905; ophthalmic surgeon to the Boston City Hospital 1901-1918; since, consulting ophthalmologist; visitng surgeon 1895-1900, visiting ophthalmic and aural surgeon since 1900, Waltham Hospital; consulting ophthalmic surgeon Massachusetts Eye and Ear Infirmary and Beth Israel Hospital (Boston), Union Hospital (Framingham), Milford Hospital (Mass.); lecturer on ophthalmology Harvard Graduate School of Medicine; professor of ophthalmology Tufts Medical School.

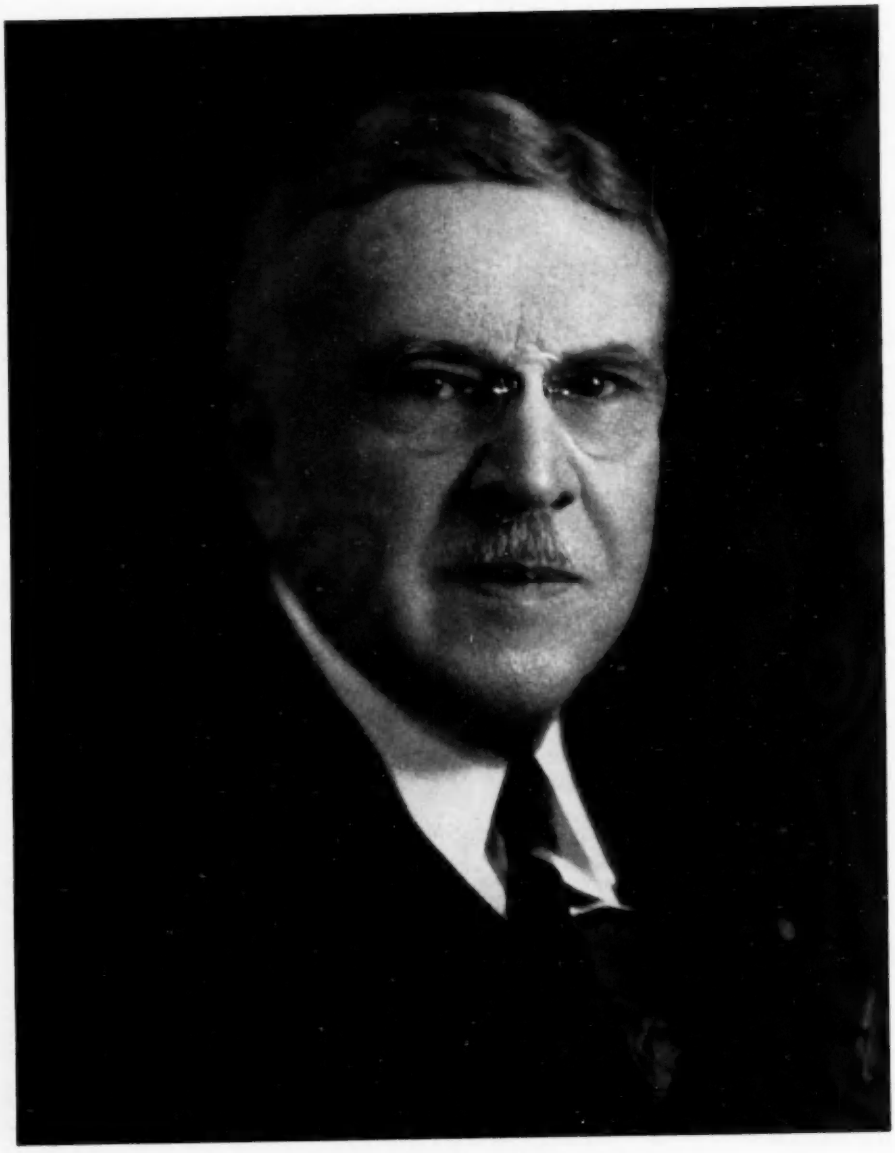
Dr. Greenwood was appointed acting assistant surgeon of the U. S. Army August 22, 1898, and was assigned to the 19th U. S. Infantry, Ponce, Puerto Rico. He was honorably discharged October 19, 1898. During the World War he was Honorary Lieutenant Colonel in the Royal Army Medical Corps with the British Expeditionary Force in France in 1916; in 1917 he became a member of the subcommittee on ophthalmology of the General Medical Board of National Defense and was on duty in the Surgeon General's office as Command Major in the Medical Reserve Corps. He was made Lieutenant Colonel in the Medical Corps and consultant in ophthalmology for the American Expeditionary Force in 1918; Colonel of the medical Reserve Corps, U. S. Army, in 1919. Following the War he compiled from the records of the Surgeon General's office the report on ophthalmology (American Expeditionary Forces). The Medical Department of the U. S. Army in the World War, Vol. XI, Part 2, Section IV, pp. 659-728.

He was chairman of the Section on Ophthalmology of the American Medical Association in 1919-1920; president of the American Academy of Ophthalmology and Otolaryngology in 1918; chairman of the Honor Society of the Academy in 1941; president of the New England Ophthalmological Society in 1903 and of the American Ophthalmological Society in 1942. He was a member of the Association for Research in Ophthalmology, Inc., and a fellow of the American College of Surgeons. He was a member of the Board for Ophthalmic Examinations (now the American Board of Ophthalmology) since 1921.

Dr. Greenwood died on October 18, 1942, in Miami, Florida.

W. L. BENEDICT

In Memoriam



LUTHER CROUSE PETER, M.D.
1869-1942

LUTHER CROUSE PETER, M.D.

1869-1942

Luther C. Peter, Professor Emeritus of Ophthalmology in the Graduate School of Medicine of the University of Pennsylvania since 1940, died November 12, 1942.

Dr. Peter, the son of Rev. Jacob and Harriet Jane (Crouse) Peter, was born in St. Clairsville, Pennsylvania, February 14, 1869. He received his elementary education in the schools at Manheim, Pennsylvania, attended Susquehanna University until 1888, and was graduated from Gettysbury College in 1891, where he did post-graduate work until 1892, at which time he received his M.A. degree. He then entered the University of Pennsylvania, and was graduated with the degree of Doctor of Medicine in 1894. In 1926 Gettysbury College awarded him a Doctor of Science degree, and Susquehanna University awarded him a similar degree in 1934; in 1938 he received a Doctor of Laws degree from Gettysbury College. The Phi Beta Kappa Chapter of Susquehanna University elected him to post-graduate membership in 1925.

He was married to Carrie Chrystine Moser, who survives him, on June 20, 1916. They had no children.

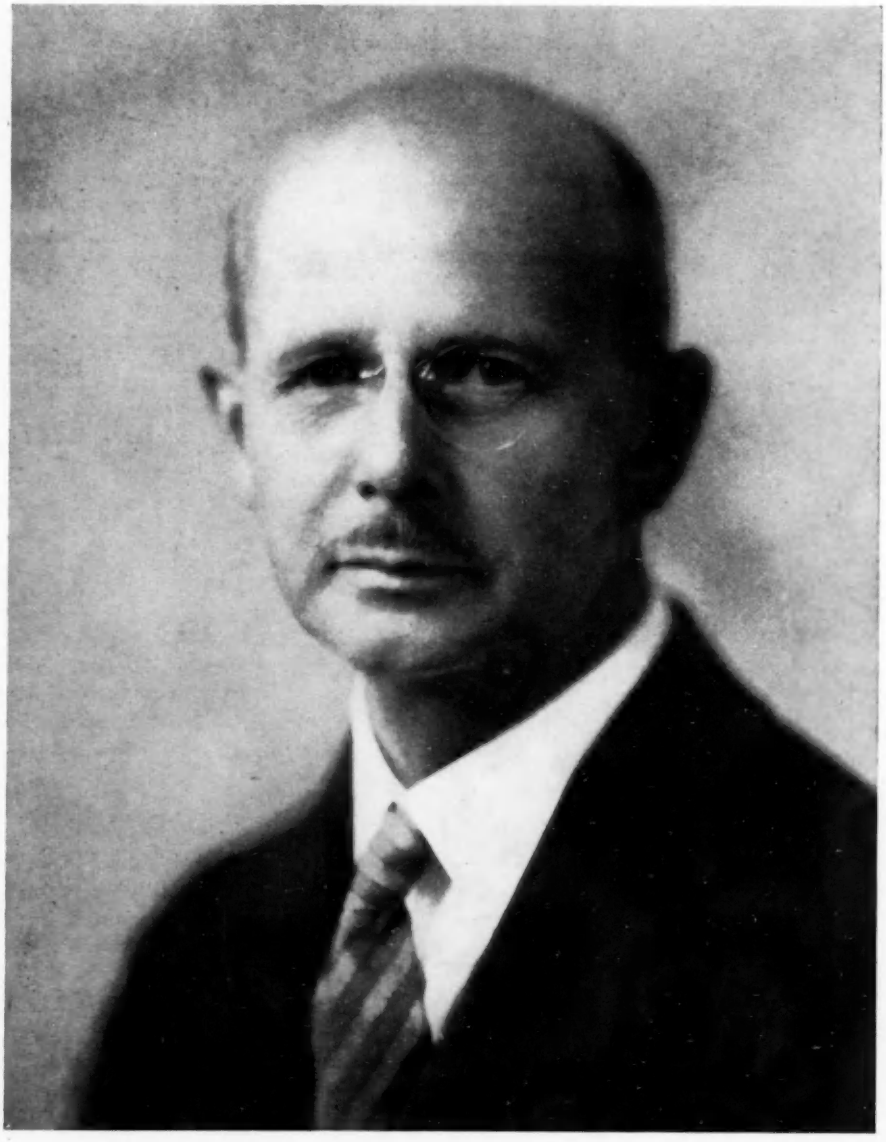
He was appointed assistant professor of Ophthalmology at the Philadelphia Polyclinic Hospital and School for Graduate Medicine in 1910, and associate professor of Ophthalmology at the same institution, in 1918, which was at that time absorbed by the University of Pennsylvania as the Graduate School of Medicine of the University, and finally, in 1919, he became professor of Ophthalmology. In 1930 he resigned from the faculty of Temple University as professor of Ophthalmology, having served in that capacity since 1917.

He was a member of the American Board of Ophthalmology from 1929 to 1937, its president 1935 to 1937; secretary of the American Academy of Ophthalmology and Otolaryngology from 1919 to 1926, and president of that Academy 1928-29; elected to the American Ophthalmological Society in 1922, to the Philadelphia College of Physicians in 1917, and to Fellowship in the American College of Surgeons in 1920. In 1922, Dr. Peter was secretary of the International Congress of Ophthalmology held in Washington, D. C.; was guest lecturer at the Oxford Congress (England) in July, 1920, his subject being "Perimetry," and again in July of 1932, this time his subject being "The Treatment of Non-Paralytic Squint."

Many articles were contributed by him to ophthalmologic literature in perimetry, disturbances of the extra-ocular muscles, in neuro-ophthalmology, the surgery of cataract and of glaucoma and more recently in the treatment of retinal separation. His book "The Principles and Practice of Perimetry" appeared in four editions, and in 1940 "The Extra-Ocular Muscles" had its third edition. While actively engaged in practice, he was on the hospital staffs, as ophthalmologist, of The Graduate Hospital of the University of Pennsylvania, Rush Hospital for Consumption and Allied Diseases, Friend's Hospital for Mental and Nervous Diseases and the Roxborough Memorial Hospital.

EDMUND B. SPAETH

In Memoriam



S. HANFORD McKEE, M.D.
1874-1942

S. HANFORD McKEE, M.D.

1874-1942

S. Hanford McKee was born in the Province of Quebec in Canada in 1874. He attended McGill University, graduating from the Medical Department in 1900. He was attracted early to the practice of Ophthalmology and underwent his training abroad. Much time was spent in London under the masters of that day, but the most intensive study was in Freiburg under the supervision of the late Professor Axenfeld. Returning to Canada, he established himself in Montreal where more than half of his time was devoted to clinics and teaching.

Early in World War I, he was commissioned in the medical forces of the Canadian Army and soon was sent overseas. At first, he served with troops in a general medical capacity, eventually commanding a Field Hospital. Later on, he was employed in his specialty. The events subsequent to the War, depressing as they were, did not destroy his enthusiasm for his chosen field of endeavor, and he continued to work long hours as clinician, teacher and practitioner.

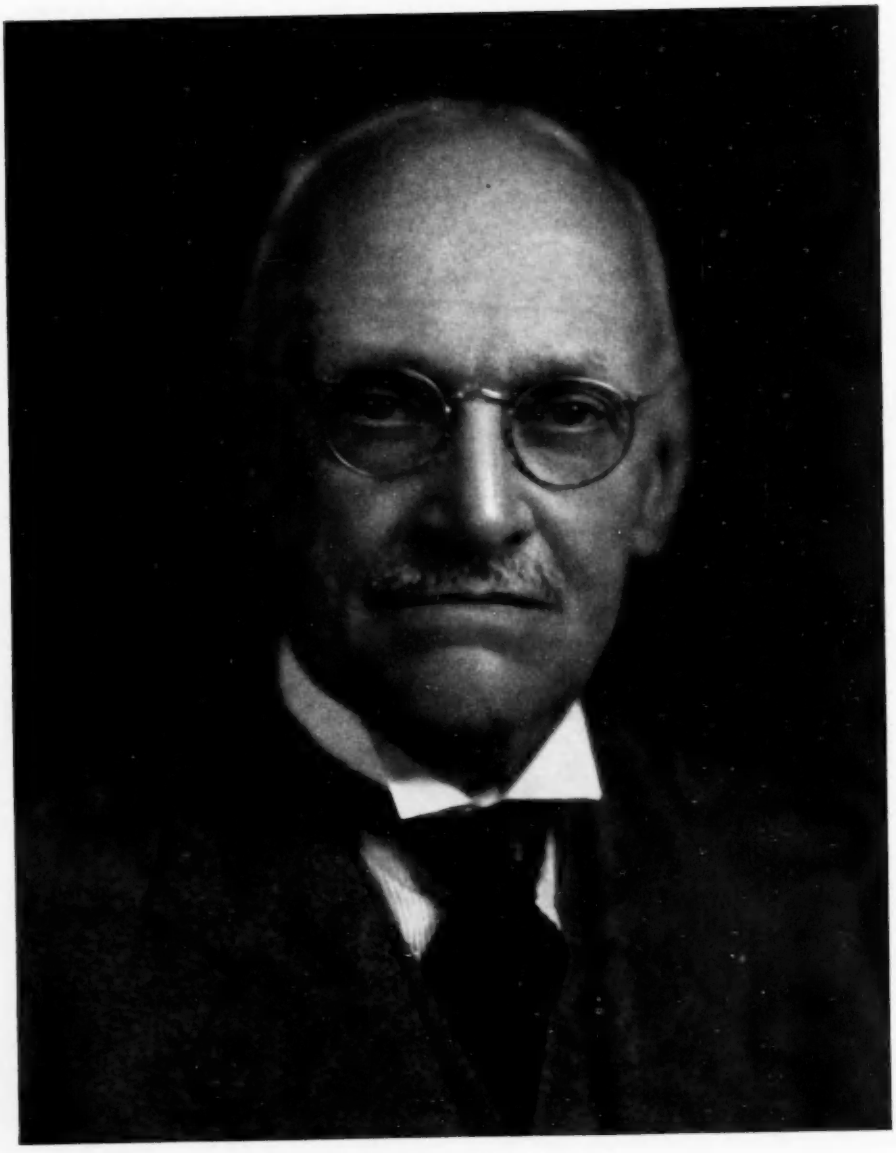
Doctor McKee was never a prolific writer, but what he did write was sound and thorough. His first major literary work was the translation and editing of Axenfeld's Bacteriology of the Eye, a massive tome that he considered re-writing only last year. Doctor McKee could be counted upon for two or three papers a year, papers that were based upon original laboratory investigations or upon clinical observations of the most meticulous type. What he wrote was worth reading.

"Han," by which name he was universally known, joined the Academy in 1908, but was not a regular attendant until immediately after the War. However, from then on, he hardly ever missed a meeting. He was in great demand in the Section on Instruction, both by the membership at large and the Committee of the Section. He gave cheerfully of his time and energy, and the men who attended his courses early recognized that he was a born teacher. He also belonged to the American Ophthalmological Society and the Section on Ophthalmology of the American Medical Association.

A wrinkling of his nose and a twinkling of his eyes attested to Han's well developed sense of humor. He loved a good story and if it was one that he thought would be useful, out would come the little note book and he would jot down a few cryptic key words. Charming as was his personality, he was of a retiring type, never seeking to intrude. His election to the presidency of the Academy was met with universal favor and acclaim for he was beloved, not alone for his work, but even more for himself.

HARRY S. GRADLE

In Memoriam



EDWARD JACKSON, M.D.
1856-1942

EDWARD JACKSON, M.D.

1856-1942

Because of his conspicuous services, his advanced age and his remarkably prolonged activity as thinker and teacher, Doctor Jackson had for years been regarded as the dean of ophthalmology. He was with us, genial and lively as ever, at the Chicago meeting October last. Two weeks after that meeting he was on his deathbed. No man has been more beloved by his colleagues, and there has been none whose passing was more widely and sincerely regretted.

Edward Jackson was born in West Goshen, Pennsylvania, March 30, 1856. Before studying medicine he had obtained the degree of Bachelor of Science in civil engineering from Union College, New York. After receiving the medical degree from the University of Pennsylvania, in 1878, he practiced general medicine for a short time at West Chester, Pennsylvania. That same year he married Jennie L. Price. He moved to Denver in 1894 on account of his wife's health, returned to Philadelphia after her death in 1896, and in 1898 again moved to Denver, where he married Emily Churchman.

In 1905 he became professor of Ophthalmology in the Medical Department of Colorado University.

In 1890 he published "Essentials of Diseases of the Eye"; in 1895, his "Skiascopy and Its Practical Application" (a profound scientific study of this objective test); and in 1900 "A Manual of the Diagnosis and Treatment of Diseases of the Eye." In addition to many hundreds of miscellaneous medical essays and editorials, he wrote the section on eye muscle operations in Wood's System of Ophthalmic Operations; that part of the chapter on ocular muscles in Wood's American Encyclopedia of Ophthalmology which deals with operations on the extrinsic muscles, and the chapter on skiascopy in the American Encyclopedia of Ophthalmology.

Doctor Jackson was president of the American Academy of Ophthalmology and Otolaryngology in 1904, chairman of the Section on Ophthalmology of the American Medical Association in 1894, and president of the American Ophthalmological Society in 1912.

At first, with financial aid from Doctors de Schweinitz and Schneideman, but chiefly on his own financial responsibility and with the burden of an annual deficit, he founded the Ophthalmic Year Book, and edited it from 1904 to 1923, inclusive. He was instrumental in creating the American Journal of Ophthalmology, third series, which took the place of a number of separate periodical publications on ophthalmology; and he edited that Journal from 1918 to 1927, inclusive. He continued to act as consulting editor of the Journal until his death, writing one or more editorials almost every month.

His most important activities were related to improvement in education for ophthalmic practice, with a plan for certification of American ophthalmologists. These efforts culminated in the creation, in 1915, of the American Board for Ophthalmic Examinations (now the American Board of Ophthalmology), which was the first organization of its kind in the United States. He was the first president of the Board and served for eleven years as one of its members. He had already persuaded the University of Colorado to follow the example of Oxford University, England, in the creation of an advanced medical degree in ophthalmology. He utilized the energy of his colleagues in the Colorado Ophthalmological Society (of which he had been one of the founders in 1899) and in the Colorado Otolaryngological Society in conducting for a number of years the Colorado Summer Course

in Ophthalmology and Otolaryngology. This enterprise pointed the way for a number of similar courses in various parts of the United States, especially the mid-winter course in Los Angeles.

Doctor Jackson was for several years on the Board of Directors of the National Society for the Prevention of Blindness, whose Leslie Dana medal he received in 1925, and he was for some years chairman of the Colorado State Commission for the Blind.

An incessant worker, Doctor Jackson placed his duty as a thinker and teacher ahead of all else. He was particularly interested in aiding the development of the younger ophthalmologists. He lived simply, even austerely, always loyal to the principles of the Society of Friends, into which he had been born.

WILLIAM H. CRISP

TRANSACTIONS
American Academy of Ophthalmology and Otolaryngology
Official Publication of the American Academy of Ophthalmology and Otolaryngology
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All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, the American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

Editorial

The Year Ahead

THE OBLIGATIONS that have been laid at the doorstep of all practitioners of medicine by the necessity of providing medical care for the civilian worker must be met by all physicians who are physically fit to carry on. War problems cannot be separated from civil problems, and military medicine becomes the universal practice.

American medical practice has become highly specialized, so much so that probably none of our thousands of physicians will claim to be equally proficient in all special fields of practice, and for some specialized fields admission of almost total ignorance is made without a blush. When one considers the high degree of proficiency in clinical practice that has been acquired by some who have devoted their entire time to a special field, technical and narrow as it may be, if by their devotion to a single objective they have been able to contribute more and more to less and less, the summation of their efforts has been to raise the level of practical medical ability to a higher level of efficiency. Yet there are no peaks in medical knowledge. The facts obtained by any one's efforts are soon disseminated through all manner of communication known to medical teachers and publishers, so that the general level of the entire professional practice is raised by a certain amount, and often enough in the past few years that raise has been considerable.

The desire of physicians to keep abreast of the times has resulted in multiple medical societies, forums of discussion, schools of instruction, libraries, laboratories, free clinics, papers and books and magazines, all maintained at a low cost, all without pecuniary gain, all for the benefit of the immense clientele that call upon the doctor for help in solution of personal problems and even public ones.

During this period of tremendous maladjustment due to the war, the need for professional advice is multiplied. Those of us who are called upon to utilize the best medical aid that the world has to give must continuously and assiduously seek ways and means of learning better methods of treatment and of increasing our efficiency in handling all methods and technics, whether old or new.

The oldest and best developed of all the medical specialties, ophthalmology and otolaryngology, have pioneered in

the fields of postgraduate instruction. Through the Academy the problem of instruction to large numbers of specialists has been solved by the annual Course of Instruction, where more than 10,000 man hours of didactic lectures are augmented by demonstrations, symposiums, panel discussions, scientific papers and visual instruction by motion pictures in the course of an intensive four or five day program. And another very important feature of the annual convention, somewhat different from that seen at other medical conventions, is technical instruction given by exhibitors of technical instruments and equipment. Instruction in the use and care of diagnostic equipment and therapeutic apparatus is as essential to a physician as is instruction to a soldier in the care and use of a rifle. Exhibitors at the Academy convention are primarily instructors rather than salesmen. Time and place for technical instruction is provided every exhibitor, and exhibitors are chosen for their usefulness to members of the Academy rather than for their financial contribution to help pay the cost of the convention. A meeting without technical exhibitors would be like a meeting without motion pictures and lantern slides. All are essential, particularly now in these urgent days.

The Academy has scored a remarkable success in the establishment and conduction of the first Home Study Course in medical specialties, a service available to qualified practitioners of ophthalmology and otolaryngology regardless of membership in the Academy. No where can so much supervised instruction in medicine be obtained at such low cost. This is made possible by the voluntary participation of more than one hundred members of the Academy whose one purpose is to help their fellow practitioners to bear the burdens of an increasing civilian and military practice. The influence of this altruistic Academy function is plainly seen as the brightest light in contemporary medical continuation study. Continuous supervised study to the individual is energized by the one convention week of intensified group instruction. The methods of instruction are complementary — all are essential for a well-rounded program such as is needed today. Attainment of the objective, however, depends upon uninterrupted operation. Once the torch is dropped, the light goes out.

W. L. BENEDICT

Dues for 1943

Dues for 1943 are payable January first. In the latter part of December notices of dues payable will be mailed to each and every member. It is probable that some members besides those in military service have recently changed their address and will not receive the notices or their delivery will be delayed.

The Council has considered suggestions that have been received from several sources that dues of members in military service be suspended for the duration of that service. An informal canvass of members in the service indicated that opinions were about equally divided. Some had been informed that dues would be suspended for the duration, but that decision was not made by the Council and when brought to the Council for action it was not sanctioned.

Included in the notice of annual dues is an amount ear marked for subscription to the TRANSACTIONS, the official organ of the Academy. In order to comply with postal rates a known paid subscription list must be furnished. The subscription must be paid by bona fide subscribers and not from the general fund of the Academy. The subscription price of the Transactions to members is $3.00, which by the way does not quite cover the cost. Consequently, the Council has decreed that dues for 1943 of Junior members who are on active military duty in the armed forces of the

United States or her allies on January 1, 1943, be reduced from $10.00 to $5.00; dues of Senior members will remain at $5.00, and Life members will be exempt from payment of dues. This applies to those who were previously informed that dues of members in military service would be suspended for the duration of that service.

W. L. BENEDICT

Committee Reports

This issue of the TRANSACTIONS contains a report of the annual Business Meeting. Included are the reports of joint and standing committees, all of which contain important information on Academy activities which are of interest to every member. These reports have been considered by the Council, and recommendations were made at the Business Meeting. Read these committee reports, write your impression to the chairman or to a member of the committee, or send a letter to the editor of the TRANSACTIONS.

W. L. BENEDICT

Notice

The American Academy of Ophthalmology and Otolaryngology offers to publish as a supplement to its TRANSACTIONS reports of completed research of interest to ophthalmologists and otolaryngologists that are too extensive for publication in current journals. Reports are to be submitted to the Editorial Board. The Academy may offer a cash award to the author in addition to publication of the research.

Reports of completed original research in the basic sciences or in the clinical field of medicine of interest to ophthalmologists and otolaryngologists may be submitted to Dr. William L. Benedict, Editor of TRANSACTIONS, 239 One Hundred First Avenue Building, Rochester, Minnesota.

The William P. Wherry Memorial

THE COUNCIL of the American Academy of Ophthalmology and Otolaryngology has authorized the development of the William P. Wherry Memorial according to the plans suggested by the Committee appointed for that purpose.

The Committee considered various characters of memorials for our late beloved Secretary, and it finally came to the conclusion that Doctor Wherry would have preferred to have his memory perpetuated in such a manner that the things in which he was vitally interested would benefit. So the Committee proposed as a memorial

A WILLIAM P. WHERRY
LIFE MEMBERSHIP IN THE ACADEMY

In brief the plan adopted is:

1. The establishment of a fund by voluntary contributions from fellow members of the Academy, as well as from friends who are not members of the Academy.
2. The use of that fund to purchase War Bonds.
3. The interest on the War Bonds to be used as payment to the Academy of initiation and Life membership dues for one person each year.
4. The recipient of the Life membership to be selected annually by the American Board of Otolaryngology as the outstanding certificant of the year.

The amount necessary for this memorial is sixteen thousand dollars. It is hoped to raise this money by voluntary contributions of five dollars or more from each member of the Academy and friends of the late Doctor Wherry. This amount will then be used to purchase Series "G" War Bonds which will bear interest in the amount of three hundred and twenty dollars annually. The amount of interest will be sufficient to pay the initiation fee (twenty dollars)

together with the annual dues (ten dollars) for thirty years.

Upon the American Board of Otolaryngology will devolve the duty of selecting the outstanding candidate from among those certificated each year. It is contemplated that such selection will be based upon a point of view that embraces all aspects of the certificants. Neither training, nor ability to pass an examination, nor clinical acumen, nor character, alone, shall be the determining factor. Rather, the recipient of the Life membership shall be the certificant, who, in the opinion of the members of the Board, is the man who most nearly approaches the plane of the ideal otolaryngologist.

As is well known to all of you, Doctor Wherry's interests lay in the Academy, the American Board of Otolaryngology and the training of young men in the *otolaryngological* specialties. These, combined with the patriotic desire of all to contribute to the war effort by buying War Bonds, seems to render this the ideal plan as a memorial to one whose life was bound up in these works.

The William P. Wherry Memorial Committee
HARRY S. GRADLE, M.D.
DEAN M. LIERLE, M.D.
DERRICK T. VAIL, M.D.

Home Study Courses

August 15th

Please underscore this date, for that means the close of registration for the Home Study Courses. We were under the impression that sufficient emphasis had been placed upon the fact that the method of conducting the Home Study Courses is such that registrations could not be accepted at any time throughout the year. But apparently we were mistaken, for requests for registration continue to flood the office. So please read carefully: *No registrants can be accepted for the Home Study Courses for the season of 1942-1943.* The next course will start on September 1, 1943, and for that registrations will be accepted up to August 15, 1943. But registrations for 1942-1943, the present courses, are closed and no additional registrants can be accepted.

At the meeting of the Council in October this Committee was authorized to initiate the Clinical Home Study Course when possible. With so many possible faculty members in the armed services, it is obviously out of the question to start the Clinical Home Study Course at present. But this Committee is prepared to initiate that course after the present emergency is past and the necessary faculty members have returned to civilian practice.

When the Clinical Home Study Course is opened it will be available to all members of the Academy and to non-members who have completed the basic Home Study Course. The clinical course will, as its name implies, consist of clinical discussions that will involve reading not only standard text books but current literature as well; and will involve answers testing the clinical deductive capabilities of the registrants. The course will run throughout the year.

Please Do Not:

1. Try to register for the 1942-1943 basic Home Study Course now.
2. Try to register for the Clinical Home Study Course that has not yet been started.

Please Do:

Register before August 15, 1943, if you wish to take the 1943-1944 basic Home Study Course in either ophthalmology or otolaryngology.

COMMITTEE ON HOME STUDY COURSES

Motion Picture Programs

October 11-14, 1942

Palmer House — Chicago

Otolaryngology

F. E. LeJeune, M.D.
Presiding

The Otolaryngological Motion Picture Program was unique in character this year. Compared to those of other years, this was different as there was only one film whch showed any operative procedure.

Dr. Dean M. Lierle's film on "Oral Lesions" was the first to be shown. It revealed unusual pathological conditions found in the mouth. The cases presented showed wise selection, and the pictures demonstrated how valuable and helpful kodachrome movies can be in teaching.

It takes time, patience and a specially devised apparatus to picture the postnasal region. Having devised the mechanism for this work himself, Dr. S. Eugene Dalton last year presented a film on "Anatomy of the Epipharynx." This year he showed pictures of various diseases found in this region. He plans to continue this work and hopes to make a film that can be set to sound.

Because of his preëminent skill and enthusiasm in bronchoscopic photography, there was no hesitancy on the Academy's part in asking Dr. Paul H. Holinger to prepare a film on "The Infant Larynx." He and his associates, Drs. Albert H. Andrews, Jr., and Ralph G. Rigby, did what was never done before. They performed an unusual and almost impossible task in making a colored movie of the *Infant Larynx*, which illustrated not only the normal infant larynx but also "congenital anomalies and acquired lesions responsible for hoarseness and dyspnea in infants."

Imagination, ingenuity and long hours of arduous work were needed to produce the film on "The Sound Conduction Apparatus in the Human Ear." This picture, made by Drs. H. G. Kobrak, J. R. Lindsay and H. B. Perlman, demonstrates what can be done when men set themselves to a task. For the first time a picture has been made showing "the acoustic movements of the ear drum of normal ears and also of ears of patients suffering from deafness."

Everyone who has tried to explain the "Technic of Vestibular Tests" to medical students knows how confused they become. Dr. J. Kent Leasure's film on that subject graphically portrays the various tests and the reactions produced. This film should be edited and set to sound for use in medical schools.

For some years Dr. Samuel J. Kopetzky has been working on sound films. Three stand to his credit — (1) Otoscopy; (2) Mastoiditis; (3) Sinus Thrombosis. These three films are especially valuable for teaching. Beginning with otoscopy, one can see pictures of various pathological lesions and follow the development step by step from myringitis to otitis media, mastoiditis and lateral sinus thrombosis. The various operative procedures in each condition are shown (in colors) and explained. This last film is Doctor Kopetzky's best. He has set a high mark for sound motion picture films for teaching. The time is ripe for the making of more sound pictures in the otolaryngological field.

John L. Myers, M.D.
Secretary

Ophthalmology

F. J. PINKERTON, M.D.
Presiding

The first film consisted of one reel showing the peeling of a corneal scar by Dr. George P. Landegger, Los Angeles. The film is in color and demonstrates the Meyer Wiener technique which consists essentially of a crucial incision across the cornea with each quadrant dissected back from the apex toward the base.

Next, an unlearnable letter chart was presented by Dr. James E. Lebensohn, of Chicago. This is designed for visual rating, primarily in the Navy, but it is also suitable for industry. Its characteristics are like that of the Grow chart. It consists of 20/20 size letters but (a) the letters are serified, (b) only the 10 letters most suitable for Snellen presentation are used, (c) each line vertically and horizontally contains these same 10 letters in different arrangements, (d) the lines are numbered to facilitate testing, and (e) the chart is incorporated in a pyralin panel to render it washable and permanent.

Next, a two-reel film showing cataract surgery was presented by Dr. A. D. Ruedemann, of Cleveland. The outstanding feature of these films is the use of animated illustrations to clarify certain points in the technique. It demonstrates that the animated pictures may have a very definite place in motion picture presentations.

The next was a two-reel film showing the surgical correction of cicatricial lagophthalmos by Dr. Edmund B. Spaeth, of Philadelphia.

The next film was one showing the O'Connor Cinch Operation by Dr. Roderic O'Connor, of San Francisco. This shows the steps in his technique very clearly.

The next film showed the O'Connor Transplantation Operation by Dr. Roderick O'Connor.

The next was a single reel illustrating dacryocystorhinostomy as done by Dr. Alton V. Hallum, of Atlanta. This film is in color and shows exceptionally well the steps of the operation. Considering the deep-lying structures that have to be viewed, the film is quite satisfactory. The operation is performed on a broad-nosed Negress, and the lacrimal fossa therefore presents itself for photographing better than it would in a person of the Caucasian race.

The next two films, which depicted exceptionally good photography, illustrated many intracapsular cataract extractions as done by Dr. Frederick A. Davis, of Madison, Wisconsin. The films illustrate primarily the fact that Doctor Davis believes in very little pull by the capsule forceps. The capsule forceps are used merely to fix the lens, and the zonules are ruptured by pressure at the limbus below.

The next was a reel showing the plastic repair of a symblepharon by Dr. Ramon Castroviejo, of New York. The photography is excellent, and the principle involved in the repair is original.

The next film was one of Dr. Ramon Castroviejo's, and it showed the surgical correction of ptosis utilizing the Wheeler procedure. The photography in this film is exceptionally good.

The last was a reel showing postoperative cicatricial strabismus and experimental and clinical considerations of the transplantation of Tenon's capsule by Doctors Berens, Romaine and Sobacke, of New York City.

ALGERNON B. REESE, M.D.
Secretary

Scientific Instruments

Presented at the Forty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology

OCTOBER 11-14, 1942 — CHICAGO

* * *

NOISE EXCLUDING DEVICE

HORACE NEWHART, M.D.
MINNEAPOLIS

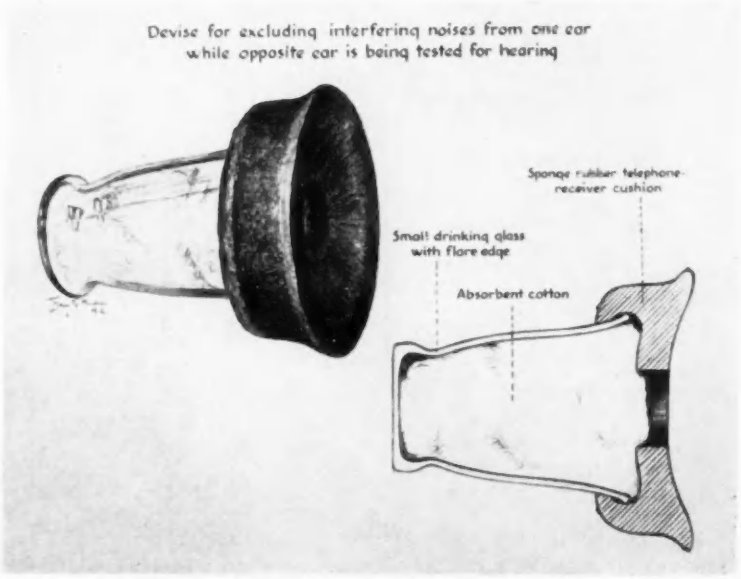

Devise for excluding interfering noises from one ear while opposite ear is being tested for hearing
Sponge rubber telephone receiver cushion
Small drinking glass with flare edge
Absorbent cotton

Figure illustrating construction of noise excluding device.

IN MAKING hearing tests in the most quiet rooms available in the average office or school building extraneous noises often prevent accuracy. In attempting to minimize the effect of such noises I recently improvised a simple, practical, inexpensive means for appreciably reducing their loudness in the opposite ear while testing the other with the pure tone audiometer, whisper, conversational voice, watch and acoumeter. It is especially useful for avoiding inaccuracy due to crossed audition when there is a considerable difference in the acuity of the two ears. The device excludes test tones of moderate intensity from the better ear.

This device was also demonstrated at the 75th Annual Meeting of the American Otological Society, Atlantic City, New Jersey, May 29, 1942.

This device consists of a No. 660 three and one-half inch Trimm soft, sponge rubber ear cushion mounted on a small fruit juice drinking glass having a flaring rim which affords a tight fit between the glass and the rubber. The glass, packed with absorbent cotton, serves as a sound insulating, non-resonating handle. The compressible ear cushion with its concave surface completely covers the auricle and when held with moderate pressure against the head acts as a tight fitting gasket. Its application materially reduces the intensity of noises which can reach the ear not under test while the other is being tested. The acoustic transmission loss is from 10 to 35 decibels depending on the predominant pitch.

It should be noted that sounds or noises of low frequency do not seriously mask test tones of high pitch.

The application of this procedure has proved more effective and cleanly than the use of the moistened finger tip or obturators placed in the auricular canal.

This device has been satisfactorily used for several months in private practice and in testing large groups of school children.

Unfortunately under present government regulations the sponge rubber ear cushions are temporarily off the market.

AN IMPROVED UNLEARNABLE LETTER CHART

(Copyright, 1943, Dr. James E. Lebensohn)

JAMES E. LEBENSOHN, M.D., PH.D.
CHICAGO

1 2 3 4 5 6 7 8 9 0
1 P H C D E T O F N Z 1
2 N E F H D C P T Z O 2
3 F P T E C Z D N O H 3
4 Z F D T N O H P E C 4
5 H Z E F T D C O P N 5
6 C D Z O F P N H T E 6
7 O C N P Z F T E H D 7
8 T O H N P E Z D C F 8
9 E N P C O H F Z D T 9
0 D T O Z H N E C F P 0
1 2 3 4 5 6 7 8 9 0

PRECISION charts with dots or gaps are virtually unlearnable but have gained only a very restricted acceptance. Letter charts have a practical appeal in that they need no preliminary explanation and permit simple answers capable of rapid checking. Their popularity continues although ophthalmologists have protested periodically for the past 70 years against their suitability as test characters.

The unlearnable letter chart devised in 1910 by Grow, a naval medical officer, has been in official use by the U. S. Navy since. It contains a series of lines of 20/20 letters. If the indicated line is not discriminated at 20 feet, the applicant slowly approaches the chart until he can respond adequately. An unlearnable letter chart requires this procedure which has the incidental advantage of provid-

From the Department of Ophthalmology, Northwestern University Medical School.

Chart is published by Boll and Lewis Optical Co., 8 S. Michigan Ave., Chicago.

ing a continuous transition in the size of the letters tested.

In the hope of improving upon the Grow design a chart of mine is submitted which differs from that of Grow's in several significant modifications:

1. The letters are serifed as only this style can approximate the Snellen standard of one minute detail in both space and line. By adhering to this principle the characters are more likely to become unrecognizable quickly when not seen clearly. The Gothic letters of the Grow chart tend to overestimate acuity somewhat so that a rate of 20/20 thereon may not grade as well under the more rigorous requirements of serif letters.

2. Of the letters authoritatively considered acceptable for Snellen presentation the 10 that seem most suitable were selected. Their gradation in difficulty according to the sequence determined by Jackson is: T C O F P D Z N E H. Among the characters deliberately omitted are L, the easiest of the Snellen letters; and B, the most difficult.

3. Each line horizontally and vertically has these same 10 letters in different arrangements. The 4 directions in which the chart may be read thus allows 40 possibilities of selection, and whatsoever line is chosen, the visual task is identical. This is certainly not the case with the Grow chart now in use.

4. The 10 letter line is a logical unit for timing the speed of vision. The applicant should read the line at the distance that determines his visual rating within 2 to 3 seconds.

5. The lines are numbered — above and below; right and left — to facilitate testing where help is limited and an exposure mechanism is not available.

6. Instead of conventional cardboard the format adopted in the Lebensohn Near Vision Chart is used. With the rugged pyralin-plywood construction, the chart has the additional advantages of being stainless, washable and permanent.

Council Minutes

SPECIAL MEETING

JUNE 28, 1942
Palmer House — Chicago

Present:

Ralph I. Lloyd, Secord H. Large, Erling W. Hansen, William L. Benedict, Derrick Vail, Frank R. Spencer, A. D. Ruedemann, Dean M. Lierle, O. E. Van Alyea, John L. Myers, James A. Babbitt, Carl H. McCaskey, Harris P. Mosher, George M. Coates, Walter H. Theobald, Frank T. Brawley.

A special meeting of the Council was called by President Ralph I. Lloyd, to be held in the Palmer House, Chicago, June 28, 1942.

The meeting was called to order by President Lloyd, who stated that its purpose was to elect an Executive Secretary-Treasurer to succeed Dr. William P. Wherry, deceased.

The Executive Committee, Dr. George M. Coates, Chairman, retired for the purpose of nomination. The Committee's report was given by Dr. Secord H. Large, nominating Dr. Wm. L. Benedict Executive Secretary-Treasurer. Doctor Large moved the adoption of the report. The vote was unanimous in favor of adoption, Doctor Benedict not voting.

No other business was introduced.

The meeting adjourned.

FORTY-SEVENTH ANNUAL SESSION*

OCTOBER 11-14, 1942
Palmer House — Chicago

SUNDAY, OCTOBER 11, 1942

Present:

James A. Babbitt, Wm. L. Benedict, Frank E. Brawley, George M. Coates, Erling W. Hansen, F. E. LeJeune, Dean M. Lierle, Ralph I. Lloyd, Carl H. McCaskey, John L. Myers, A. D. Ruedemann, Walter H. Theobald, O. E. Van Alyea.

I

The meeting was called to order by President Lloyd at 9:30 a.m.

II

The Executive Secretary presented communications for Council consideration.

1. A letter from Dr. Burt R. Shurly, Chairman of a Special Committee on National Defense, was presented. (See Activities 1-6, 1941 — continuous committee.)

It was moved and seconded that the Committee be discontinued. Motion carried. (See Council Action XV.)

4. The President proposed and the Council recommended by acclamation that Dr. W. L. Benedict act on all committees in place of Dr. W. P. Wherry, deceased.

5. A communication was received from Dr. Harry S. Gradle and Dr. Jonas Friedenwald suggesting the establishment of a Standing Committee for the Standardization of Tonometers. The following Committee was suggested:

Francis H. Adler, Philadelphia
Jonas S. Friedenwald, Baltimore
Adolph Posner, New York City
Sanford R. Gifford, Chicago
Peter Kronfeld, Chicago
Harry S. Gradle, Chicago
D. O. Harrington, San Francisco

It was moved and seconded that the recommendations be approved and that the Committee suggested by Drs. Gradle and Friedenwald be appointed.

6. A letter from Dr. Ralph Rychener tendering his resignation as Assistant Secretary for Ophthalmology was presented.

It was moved and seconded that the resignation of Dr. Ralph Rychener be accepted with regret. The Secretary was instructed to prepare a letter to be signed by the officers expressing their regret to Doctor Rychener.

8. A communication from Joseph P. Hackel, regarding the exhibition of especially prepared films on medical subjects, was presented. The communication was referred to the Executive Committee with the suggestion that the matter of production of motion pictures be left in the hands of the Board of Secretaries. (See Council Action XII — 1.)

9. Diversion Club. It was suggested by Dr. Carl H. McCaskey, who is Chairman of the Diversion Club, that the activities of the Diversion Club be extended to include entertainment features connected with the convention activities. It was also suggested that the activities of the Diversion Club be sub-

* Some items are omitted from this report. Complete Minutes are on file in the office of the Executive Secretary-Treasurer.

sidized to a reasonable extent from Academy funds.

Referred to the Executive Committee.
(See Council Action XII — 2.)

III

Report of Executive Secretary

1. The Executive Secretary reported the removal of the executive office from Omaha to Rochester on September 7, 1942. Miss Clare McGovern from the Omaha office and Miss Frieda Hess moved to Rochester to continue work with the Executive Secretary.

2. Membership:

Junior	2,854
Senior	164
Life	202
Honorary	17

3. Applications from 208 candidates have been received. The names have been published in the TRANSACTIONS according to the By-Laws.

Referred to the Executive Committee.
(See Council Action XII — 4.)

4. Resignations have been received from 10 members.

Frederick L. Beck, Cheyenne, Wyoming
James G. Calhoun, St. Louis, Mo.
Frank E. Detling, Los Angeles, Calif.
G. D. Hallett, New York, N. Y.
A. E. Johnson, Watertown, S. D.
Phillip Mysel, Boston, Mass.
Clarence Rainey, Wilmette, Ill.
D. L. Shumate, Kansas City, Mo.
Robert B. Sweet, Long Beach, Calif.
Robert E. Wright, Dallas, Texas

Referred to Executive Committee.
(See Council Action XII — 3.)

5. Members deceased — 46.

Herbert Stanley Birkett, Montreal, Canada
Melville Black, Denver, Colorado
Alice Gertrude Bryant, Boston, Mass.
Robert Emmett Buckley, New York, N. Y.
John DeWitt Byrne, DuQuoin, Illinois
Donald M. Campbell, Detroit, Michigan
Ernest Willoughby Carpenter, Greenville, S. C.
George Washington Carr, Englewood, N. J.
Ralph Livingston Daniels, New Bern, N. C.
Harry B. Davis, Kansas City, Mo.
Dred Rumley Dorente, Ada, Oklahoma
Louis S. Dunn, Philadelphia, Pa.
William A. Ellingwood, Rockland, Maine
C. C. Eves, Philadelphia, Pa.
William Fowler, Detroit, Mich.
Henry Wm. George, Middletown, Pa.
Aaron S. Green, San Francisco, California
J. Beatty Griffith, Washington, D. C.
Emile de Grosz, Budapest, Hungary
Philip Allen Halper, Chicago, Ill.
Max Wm. Jacobs, St. Louis, Mo.
Charles Joseph Jaquish, Houston, Texas
Casper Walker Jennings, Greensboro, N. C.
John Joseph Killeen, Chicago, Ill.
Benjamin F. Macnaughton, Montreal, Canada
Carl Charles McClelland, Detroit, Mich.
Richmond McKinney, Memphis, Tenn.
John Oliver McReynolds, Dallas, Texas
Stanley G. Miner, Detroit, Michigan
Wm. T. Morrissey, New Britain, Conn.
Edward Powers Norcross, Highland Park, Ill.
Henry Louis Pollock, Chicago, Ill.
Wm. Aubrey Poole, Lexington, Ky.
Winfred Bryant Post, Carthage, Mo.
John Abraham Pratt, Minneapolis, Minn.
Robert Grisham Reaves, Knoxville, Tenn.
George S. Row, Indianapolis, Ind.
Edward Raymond Ryan, Milwaukee, Wis.
Joseph J. Shafer, Louisville, Ky.
Francis Wayles Shine, New York, N. Y.
John Lee Summerlin, Gainesville, Florida
Jesse L. Wagner, Reading, Pa.
Nelson Shelly Weinberger, Sayre, Pa.
Wm. P. Wherry, Omaha, Nebraska
Casey Albert Wood, Pasadena, California
W. Stuart Woodruff, Colebrook, Conn.

6. The Executive Secretary recommended that Miss Clare McGovern be given the title Registrar. A motion was made by Dr. Walter H. Theobald, seconded by Dr. Carl H. McCaskey that Miss Clare McGovern be made Registrar of the Academy. Motion carried.

7. The Executive Secretary suggested that eligibility for membership in the Academy be extended to include specialists in esophagoscopy, bronchosopy and oral and plastic surgery who have been certificated by their respective Boards, but who may not hold a certificate of the American Board of Otolaryngology. This was referred to the Executive Committee.

(See Council Action XII — 13.)

8. The Executive Secretary brought up for discussion the question of incorporation of the Academy. Moved by Dr. Carl H. McCaskey, seconded by Dr. A. C. Snell, that the Executive Secretary be authorized to employ an attorney to investigate and report on the advisability of incorporation. Motion carried.

9. The Executive Secretary discussed the advisability of reorganizing the Editorial Board. The matter was referred to the Executive Committee.

(See Council Action XII — 8.)

IV

Report of the Treasurer

The Treasurer presented the report of the auditor for the close of the fiscal year, August 31, 1942.

(See auditor's report in the Minutes of the annual Business Meeting.)

V

Honorary Membership

Doctor Kaspar Pischel, of San Francisco, and Doctor E. C. Ellett, of Memphis, were nominated Honorary Members of the Academy.

VI

Report of Investment Committee

The roster of the Academy held securities was presented as a part of the auditor's report. The Committee has not held a meeting nor prepared a report except a statement by Doctor Burch on the present estimated value of the securities.

Moved by Dr. George M. Coates, seconded by Dr. Walter H. Theobald, that the Ex-Officio Committee on Investments be discontinued. Carried.

(See Council Action XVII.)

VII

Report of Section on Public Relations

Secretary Hansen reported on the activities of the Publicity Committee. He referred to the ruling of the Council regarding the advisability of releasing press reports on papers or other contributions to the program by members living in the city in which the convention is held. He recommended that such members not be so penalized.

Moved by Dr. Carl H. McCaskey, seconded by Dr. George M. Coates, that this restriction be removed and that the Secretary for Public Relations be permitted to use his judgment regarding all press releases. Motion carried.

Doctor Hansen suggested the appointment of an assistant secretary for Public Relations. The request was referred to the Executive Committee.

(See Council Action XII — 10.)

MONDAY, OCTOBER 12, 1942

Present:

James A. Babbitt, Wm. L. Benedict, Frank E. Brawley, J. McKenzie Brown, Geo. M. Coates, Erling W. Hansen, F. E. LeJeune, Dean M. Lierle, Ralph I. Lloyd, Carl H. McCaskey, John L. Myers, F. J. Pinkerton, A. D. Ruedemann, Albert C. Snell, Walter H. Theobald, O. E. Van Alyea.

VIII

The meeting was called to order by President Lloyd at 7:30 a.m.

IX

Reports of Standing Committees

(Full reports of Standing Committees are published in the report of the annual Business Meeting.

1. Report of American Board of Ophthalmology. Report received. Referred to Activities Committee.

2. Report of American Board of Otolaryngology. Dr. T. E. Carmody. Report received. Referred to Activities Committee.

3. Report of Committee on Ophthalmic Pathology. Dr. Harry S. Gradle. Report received. Referred to Activities Committee.

4. Report of Committee on Otolaryngic Pathology. Dr. Frank J. Novak, Jr. Report received. Referred to Activities Committee.

5. Report of Committee on Conservation of Hearing. Dr. Horace Newhart. Report received. Referred to Executive Committee. (See Council Action XII — 12.)

6. There was no report from the American Association for the Advancement of Science. President Lloyd stated that he had had a communication from the A. A. A. S. asking that a delegate be appointed to replace Doctor Wherry, deceased. Dr. W. L. Benedict was appointed by the President.

7. Report of Scientific Exhibit. Dr. O. E. Van Alyea. Report received and referred to Activities Committee.

8. Report of Liaison Committee of the National Society for the Prevention of Blindness. Dr. W. H. Luedde presented a report on the activities of this Committee. Inasmuch as a Committee with identical functions has been appointed by the Section on Ophthalmology of the A. M. A. and inasmuch as the Committee which Dr. Luedde represents for the Academy has not been active, he and Doctor Pfingst recommended the termination of this Committee. It was moved by Dr. Carl McCaskey and seconded by Dr. John L. Myers that the Committee be discontinued. Motion carried.

9. There was no report from the Committee on Ophthalmic Literature. (Dr. Conrad Berens, Chmn.) Moved by Dr. Frank Brawley, seconded by Dr. John L. Myers that the Committee on Ophthalmic Literature be discontinued.

10. Report on Home Study Courses. Dr. Harry S. Gradle. Report received and referred to Activities Committee.

Dr. Gradle proposed that the Home Study Course establish a clinical home study course:

(1) To be open to all members of the Academy and to non-members who have completed the basic Home Study Course.

(2) The course to be operated as follows: On the first of each month a complete clinical description of a hypothetical case, together with reading references of text books and recent journals, to be sent to each registrant. At the same time he will be asked to make a diagnosis differentiating from similar conditions; discuss the pathology present; outline the treatment; discuss possibilities and management of complications. His paper would then be returned for correction and finally sent back to him for final file.

(3) Doctor Gradle believes that this could be done at a cost of $15.00 a year — possibly less.

(4) Doctor Gradle proposes that the men correcting the papers be paid 50 cents a paper for the corrections and that each month's papers be corrected by as small a group as is possible.

(5) The set-up for the present Home Study Course, with some expansion, could handle the proposed clinical course.

Motion made by Dr. Walter H. Theobald and seconded by Dr. John L. Myers that the Home Study Course proceed with the inauguration of the clinical course when and if conditions appear favorable. Motion carried.

X

Report of Joint Committees

(Full reports of Joint Committees are published in the report of the annual Business Meeting.)

1. Committee on Optics and Visual Physiology. Dr. Chas. Bahn. Report received. Referred to Activities Committee.

(See Council Action XII — 11 and XIII — 7.)

2. Eye Health Committee of the American Student Health Association. Dr. Wm. L. Benedict. Report received. Referred to Activities Committee.

(See Council Action XII — 11.)

3. American Orthoptic Council. Dr. LeGrand Hardy. Report received. Referred to the Activities Committee.

(See Council Action XII — 11 and XIII — 9.)

4. Committee on Industrial Ophthalmology. Dr. John B. Hitz. Report received. Referred to the Activities Committee.

(See Council Action XIII — 10 and XXIV.)

TUESDAY, OCTOBER 13, 1942

Present:

James A. Babbitt, Wm. L. Benedict, Frank E. Brawley, J. McKenzie Brown, Geo. M. Coates, Erling W. Hansen, F. E. LeJeune, Dean M. Lierle, Ralph I. Lloyd, Carl H. McCaskey, John L. Myers, F. J. Pinkerton, A. D. Ruedemann, Albert C. Snell, Walter H. Theobald, O. E. Van Alyea.

XI

The meeting was called to order by President Lloyd at 7:30 a.m.

XII

Report of Executive Committee

1. Medical Films.

The Executive Committee recommends that no change be made in our present arrangement for handling and producing medical films.

Approved by Council consent without motion.

2. Diversion Club.

The Executive Committee recommends to the Council that the Diversion Club extend its activities to include all entertainment features in connection with the convention of the Academy.

(See Council Action II — 9.)

Unanimously approved by Council consent without motion.

3. Resignations.

The Committee recommended that the resignations of the 10 members mentioned in Council Action III — 4 be accepted.

Unanimously approved by Council consent without motion.

4. Candidates for membership.

(See list published in report of annual Business Meeting.)

5. Dues.

A) Delinquent dues.

The Executive Committee recommends that the Executive Secretary-Treasurer make all reasonable effort to collect delinquent dues: the Secretary was instructed to inform members whose dues are delinquent for five years or more that they have been dropped from membership.

Unanimously approved by Council Action.

B) Annual dues.

The Executive Committee recommends that Junior members in active military service on January 1, 1943, pay one-half the regular dues for the calendar year 1943.

Unanimously approved by Council Action.

8. The Editorial Board. The Executive Committee recommends that no immediate change in the personnel of the Editorial Board be made. It was suggested that the Editor of the Transactions be empowered to obtain whatever editorial help is necessary for immediate requirements.

Unanimously approved by Council Action.

10. Nomination of officers.

President-Elect — Dr. Lawrence T. Post, St. Louis, Mo.

1st Vice President — Dr. Sanford R. Gifford, Chicago, Ill.

2nd Vice President — Dr. Wesley Bowers, New York, N. Y.

3rd Vice President — Dr. Wm. Krieger, Poughkeepsie, N. Y.

Comptroller — Dr. Secord H. Large, Cleveland, Ohio

Executive Secretary-Treasurer — Dr. W. L. Benedict, Rochester, Minn.

Secretary for Otolaryngology — Dr. O. E. Van Alyea, Chicago, Ill.

Secretary for Ophthalmology — Dr. Algernon B. Reese, New York, N. Y.

Secretary for Instruction — Otol. — Dr. Dean M. Lierle, Iowa City, Iowa

Secretary for Instruction — Ophth. Dr. A. D. Ruedemann, Cleveland, Ohio

Secretary for Public Relations — Dr. Erling W. Hansen, Minneapolis, Minn.

Member of Council (1946) — Dr. C. H. McCaskey, Indianapolis, Indiana

Liaison Officer, Honor Society — Dr. E. C. Ellett, Memphis, Tenn.

(Nomination of Assistant Secretary for Public Relations deferred.)

Dr. J. M. Brown moved that the nomination of officers be accepted. Dr. Carl H. McCaskey seconded the motion. Carried.

11. Nominations to Standing and Joint Committees.

To represent the Academy on the American Board of Ophthalmology — Dr. Algernon B. Reese, New York, N. Y.

To represent the Academy on the American Board of Otolaryngology — Dr. Frank R. Spencer, Boulder, Colorado.

Ophthalmic Pathology — Dr. Harry S. Gradle, Chairman; Drs. F. H. Verhoeff, J. S. Friedenwald and Georgiana Dvorak-Theobald.

Otolaryngic Pathology — Dr. Frank J. Novak, Jr., Chairman; Drs. Herman Z. Semenov and LeRoy A. Schall.

Committee on Conservation of Hearing — Dr. Dean M. Lierle, Chairman.

The American Association for the Advancement of Science — Dr. W. L. Benedict; Dr. Harry S. Gradle, official delegate.

Scientific Exhibit — Dr. O. E. Van Alyea, Chairman; Dr. Georgiana Dvorak-Theobald, Consultant.

Home Study Courses — Dr. Harry S. Gradle, Ophthalmology; Dr. Frank J. Novak, Jr., Otolaryngology; Dr. Daniel Snydacker, Ophthalmology; Dr. L. Benno Bernheimer, Otolaryngology.

American Committee on Optics and Visual Physiology — Dr. Charles A. Bahn, Chairman; Drs. A. D. Prangen, Alfred Cowan.

Eye Health Committee of the Student Health Association — Dr. Lawrence T. Post.

American Orthoptic Council — Drs. A. D. Prangen, John B. Hitz, Murray B. McCaslin.

Committee on Industrial Ophthalmology — Drs. John B. Hitz, Thomas D. Allen, Edwin B. Dunphy.

Nominations accepted by the Council.

12. The Executive Committee recommends to the Council that Dr. Dean M. Lierle be appointed Chairman of the Committee on Conservation of Hearing with power to reorganize it. It further recommends that $1,000.00 be appropriated for use of the Committee for whatever purpose the chairman may direct, the amount to be paid on warrant supplied by the chairman. (See Council Action XIV — 3.)

13. The Executive Committee recommends to the Council that the eligibility requirements for membership in the Academy be altered so as to permit the election to membership of maxillo-facial surgeons who have been certificated by the American Board of Plastic Surgery. Approved by Council Action.

14. The Executive Committee voted to hold the 1943 convention at the Palmer House, Chicago. Approved by Council Action.

XIII

Report of Activities Committee

(Committee reports are published in the report of the annual Business Meeting.)

1. Report of American Board of Ophthalmology. Received.

2. Report of American Board of Otolaryngology. Received.

3. Report of Registry of Ophthalmic Pathology. Approved and recommended adoption.

4. Report of Registry of Otolaryngic Pathology. Approved and recommended adoption.

5. Report of Scientific Exhibit. Received.

6. Report of Home Study Courses. Approved and recommended adoption.

(See Council Action IX — 10.)

7. Report of American Committee on Optics and Visual Physiology. Approved and recommended adoption. (See Council Action XIV — 4.)

The Council adopted the recommendation of the Committee on Optics and Visual Physiology that the Keystone View Company be permitted to exhibit at the Academy convention.

8. Report of Eye Health Committee of American Student Health Association. Received.

9. Report of Orthoptic Council. Approved and recommended adoption. (See Council Action XIV — 2.)

10. Report of Committee on Industrial Ophthalmology. Approved and recommended adoption.

(See Council Action XXIV.)

XIV

Report of Finance Committee

1. American Registry of Pathology of the Army Medical Museum.

The Finance Committee recommends payment of a deficit of $73.75 incurred by the American Registry of Pathology of the Army Medical Museum in 1942. It further recommends that $1,500.00 be appropriated for the use of the Registry for 1943. Approved by Council Action.

2. American Orthoptic Council.

The Finance Committee recommends that an appropriation of $100.00 be made to the Orthoptic Council for 1943. Approved by Council Action.

3. Committee on Conservation of Hearing.

The Finance Committee recommends the appropriation of $1,000.00 toward the work of the Committee on Conservation of Hearing for the year 1943. Approved by Council Action.

4. American Committee on Optics and Visual Physiology.

By mail vote, dated November 3, 1942, the members of the Finance Committee recommended the appropriation of $50.00 toward the work of the American Committee on Optics and Visual Physiology for the year 1943.

XV

Committee on War Activities

On motion by Dr. Burt Shurly, seconded by Dr. James A. Babbitt, a committee to be known as the Committee on War Activities was established. The President appointed Dr. Burt Shurly chairman with power to appoint four additional members. The President and Secretary-Treasurer are to be ex-officio members of this committee.

(See Council Action II — 1.)

XVI

Teachers' Section

The Executive Secretary advised discontinuing the Teachers' Section because the functions of this Section, as carried in the past, have largely been taken over by other committees. On motion by Dr. W. L. Benedict, seconded by Dr. George M. Coates, the Section was discontinued.

XVII

Ex-Officio Investment Committee

On motion by Dr. George M. Coates, seconded by Dr. W. L. Benedict, the Committee is discharged with thanks and the functions of the Ex-Officio Investment Committee are to be assumed by the Executive, Research and Finance Committee.

XVIII

The President recommended that the General Activities Committee be appointed by the President to include so far as is possible members of the Council who are familiar with the activities of the Standing Committees. (See Council Action 1931 — four members plus Chairman of Program Committee who shall be the Chairman.)

XIX

William P. Wherry Memorial

The Memorial Committee recommended that a paid up Life membership in the Academy be granted annually as a memorial to Dr. Wm. P. Wherry to the outstanding candidate selected by the American Board of Otolaryngology. The Life membership in the Academy amounts to $320.00 per year, including initiation fee and thirty years' dues. The Committee recommended that an appeal be made to all members of the Academy as well as to other friends of Dr. Wm. P. Wherry to donate at least five dollars each to establish a fund of $16,000. Money thus raised is to be invested in 2% series "G" War Bonds. It is estimated that the income from these bonds will be $320.00 per year; the deficit on October 1, 1943, to be made up from current funds of the Academy.

Recommended and adopted by Council Action.

XX

Motion made by Dr. John L. Myers, seconded by Dr. W. L. Benedict, that a committee be appointed to revise the constitution and by-laws.

Motion carried.

WEDNESDAY, OCTOBER 14, 1942

Present:

James A. Babbitt, Wm. L. Benedict, Frank E. Brawley, Geo. M. Coates, Erling W. Hansen, F. E. LeJeune, Dean M. Lierle, Ralph I. Lloyd, Carl H. McCaskey, John L. Myers, A. D. Ruedemann, Walter H. Theobald, O. E. Van Alyea, Harris P. Mosher, Lawrence T. Post, Wm. A. Krieger.

XXI

The meeting was called to order by President Lloyd at 7:30 a.m.

XXII

Honor Society

It was moved by Dr. Harris P. Mosher, seconded by Dr. Dean M. Lierle, that the President of the Honor Society be the Liaison Officer to the Council. Carried.

XXIII

Awards

The Committee on Awards for Scientific Exhibits this year was selected by Dr. Harris P. Mosher, with advice and suggestions from Dr. O. E. Van Alyea. All members of the Award Committee were not members of the Honor Society. The Council approved.

XXIV

Committee on Industrial Ophthalmology

Dr. A. C. Snell requested information regarding the advisability of appointing to the Committee on Industrial Ophthalmology as associate members persons who are not members of the Academy. He mentioned particularly a professor from Purdue University. It was the ruling of the chair that the officers of the Committee have power to add to their Committee persons who are not members of the Academy. Approved.

XXV

The Council directed the Executive Secretary to send a formal message to Mrs. W. P. Wherry.

The Council adjourned.

Members who anticipate changes of address are asked to send them to the office of the Executive Secretary, 239 One Hundred First Avenue Building, Rochester, Minnesota, 30 days before the mailing date of the TRANSACTIONS in order to guarantee its delivery.

Business Meeting

OCTOBER 13, 1942

Minutes

The business meeting of the Forty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, held at the Palmer House, Chicago, Illinois, Tuesday afternoon, October 13, 1942, convened at five-thirty o'clock. Dr. Ralph I. Lloyd, President of the Academy, presided.

PRESIDENT LLOYD: Gentlemen, the meeting will please come to order.

The first item is the report of the Executive Secretary.

EXECUTIVE SECRETARY-TREASURER BENEDICT: Mr. Chairman and Members: The present membership of the Academy is as follows: Honorary, 17; Life, 202; Senior, 164; Junior, 2,854; Total, 3,237 members. At this meeting there will be presented for election to membership the names of 207 candidates which will bring our total membership to 3,444.

PRESIDENT LLOYD: The report of the Treasurer.

EXECUTIVE SECRETARY-TREASURER BENEDICT: The following is the report of the Auditor which constitutes the Treasurer's report.

JOHN P. BEGLEY
Certified Public Accountant
CREIGHTON UNIVERSITY
OMAHA, NEBRASKA

September 5, 1942

The American Academy of
Ophthalmology and Otolaryngology
Omaha, Nebraska

Gentlemen:

I have examined the accounts and records of the American Academy of Ophthalmology and Otolaryngology, an unincorporated association, for the fiscal year ended August 31, 1942. My examination was made in accordance with generally accepted auditing standards applicable in the circumstances and included all procedures which I considered necessary. I did not make a detailed audit of all transactions.

Office furniture, instruments and equipment have not been capitalized.

The securities owned are in possession of the Trust Department of the Omaha National Bank for safekeeping. Investments are carried at cost, with no adjustments for amortization of premium or discount. Interest has not been accrued.

Usually the ordinary general expenses are paid in the month following the one in which they are incurred. For the reason that the Academy office is being moved from Omaha to Rochester, most items of expense for the month of August have been paid.

The changes in the financial status of the Academy during the fiscal year are accounted for as follows:

Assets held Sept. 1, 1941 (Book value)		$144,168.01
Deduct: Excess of disbursements over receipts		
Disbursements	$82,594.73	
Receipts	70,605.22	
	$11,989.51	
Loss on sale of securities	1,875.20	13,864.71
Assets held Aug. 31, 1942 (Book value)		$130,303.30

The schedule of securities owned shows the market value of the holdings August 31, 1942. The net worth of the Academy, exclusive of furniture and equipment, is $97,246.90.

Respectfully submitted,
JOHN P. BEGLEY
Certified Public Accountant

THE AMERICAN ACADEMY OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY
OMAHA, NEBRASKA

Receipts and Disbursements

September 1, 1941 to August 31, 1942

Cash balance Sept. 1, 1941		$ 19,708.78
Receipts:		
Applications, dues, etc.	$24,600.08	
Transactions	13,630.47	
Conventions	26,024.31	
Home study courses	2,540.00	
Income from securities	3,569.36	
Diversion club	241.00	70,605.22

On Capital Account:		
Securities sold	13,249.80	
		$103,563.80
Disbursements:		
Research	$ 2,745.55	
Transactions	15,237.13	
Honorariums	9,738.68	
Meetings and conventions	37,576.28	
Office and general expense	15,613.58	
Diversion club expense	5.60	
Home study courses	1,677.91	
	$82,594.73	
On Capital Account:		
Securities purchased	$ 5,947.50	$ 88,542.23
Cash balance August 31, 1942		$ 15,021.57

Summary of Assets, August 31, 1942

Cash balance	$ 15,021.57
Securities owned (at cost)	115,281.73
	$130,303.30
Cash balance	$ 15,021.57
Securities owned (market value)	82,225.33
	$ 97,246.90

Cash

Omaha National Bank, Omaha, Nebraska

Balance per bank statement, August 31, 1942	$14,131.52
Less: Wm. G. Patton item not debited	10.00
Last check issued No. 1381. Checks No. 1363, 1371, and 1380 were outstanding and were not taken into the accounts.	$14,121.52
Bank of Montreal, Toronto, Ontario. Balance per bank statement, August 31, 1942	900.05
Total Cash, August 31, 1942	$15,021.57

The American Academy of Ophthalmology and Otolaryngology

Omaha, Nebraska

Schedule of Securities Owned August 31, 1942

	Rate	Due	Par Value	Cost
American International Corp.	5½%	1949	$ 2,000	$ 1,962.50
Brooklyn Edison Co., Inc.	3¼%	1966	3,000	3,045.00
City of New York Corporate Stock for Transit Unification	3 %	1980	4,700	4,700.00
Chicago Milwaukee, St. Paul & Pacific Rr. Co. Mtg. Series A	5 %	1975	2,000	1,821.50
Chicago Rock Island & Pacific Rr. Co. 1st & Ref. Mtg.	4 %	1934	3,000	2,668.50
Chicago & Western Indiana Rr. Co.	4¼%	1962	5,000	5,100.00
The Citizens Bldg. Co. of Cleveland 1st Mtg. Leasehold S. F.	6 %	1946	5,000	5,000.00
The Citizens Bldg. Company of Cleveland 50 shares of no par stock	....	....	....	.....
The Kansas City Ft. Scott & Memphis Ry. Co. Ref. Mtg.	4 %	1936	5,000	4,352.50
Kingdom of Denmark	5½%	1955	2,000	2,020.00
Louisville Gas & Electric Co., 200 shares of 5% Cumulative Pfd. Stock par $25 par share				5,450.00
Massachusetts Investors Trust	978 shares			27,673.90
Missouri Pacific Rr. Co. 1st & Ref. Mtg. Series "G"	5 %	1978	2,000	1,934.00
Missouri Pacific Rr. Co. 1st & Ref Mtg. Series "I"	5 %	1981	3,000	2,850.00
Ophthalmic Publishing Company 5 shares of stock $20 par				Gift

Shell Union Oil Corp.	2½%	1954	5,000	4,887.50
Southern Pacific Company	4½%	1981	5,000	4,837.50
The Van Sweringen Company Certificates of Indebtedness	6 %	1948	6,000	6,031.33
Witherbee Sherman Corp 50 shares Pfd. stock $50 par (Acquired in reorganization. Bonds have been sold.)				
Union Pacific Rr. Co.	3½%	1971	6,000	5,947.50
U. S. Savings Bonds "D"	3⅓%	1949	10,000	7,500.00
U. S. Savings Bonds "D"	3⅓%	1950	10,000	7,500.00
U. S. Savings Bonds "G"	2½%	1953	10,000	10,000.00
				$115,281.75

PRESIDENT LLOYD: Necrology.

EXECUTIVE SECRETARY-TREASURER BENEDICT: I will now read the names of the members of the Academy who have passed on since our last meeting:*

Herbert Stanley Birkett, Montreal, Canada
Melville Black, Denver, Colorado
Alice Gertrude Bryant, Boston, Massachusetts
Robert Emmett Buckley, New York, N. Y.
John DeWitt Byrne, Du Quoin, Illinois
Donald M. Campbell, Detroit, Michigan
Ernest Willoughby Carpenter, Greenville, South Carolina
George Washington Carr, Englewood, New Jersey
Ralph Livingston Daniels, New Bern, North Carolina
Harry B. Davis, Kansas City, Missouri
Dred Rumley Dorente, Ada, Oklahoma
Louis S. Dunn, Philadelphia, Pennsylvania
William A. Ellingwood, Rockland, Maine
C. C. Eves, Philadelphia, Pennsylvania
William Fowler, Detroit, Michigan
Henry William George, Middletown, Pensylvania
Aaron S. Green, San Francisco, California
J. Beatty Griffith, Washington, D. C.
Emile de Grosz, Budapest, Hungary
Philip Allen Halper, Chicago, Illinois
Max William Jacobs, St. Louis, Missouri
Charles Joseph Jaquish, Houston, Texas
Casper Walker Jennings, Greensboro, North Carolina
John Joseph Killeen, Chicago, Illinois
Benjamin F. Macnaughton, Montreal, Canada
Carl Charles McClelland, Detroit, Michigan
Richmond McKinney, Memphis, Tennessee
John Oliver McReynolds, Dallas, Texas
Stanley G. Miner, Detroit, Michigan
Wm. T. Morrissey, New Britain, Connecticut
Edward Powers Norcross, Highland Park, Illinois
Henry Louis Pollock, Chicago, Illinois
William Aubrey Poole, Lexington, Kentucky
Winfred Bryant Post, Carthage, Missouri
John Abraham Pratt, Minneapolis, Minnesota
Robert Grisham Reaves, Knoxville, Tennessee
George S. Row, Indianapolis, Indiana
Edward Raymond Ryan, Milwaukee, Wisconsin
Joseph J. Shafer, Louisville, Kentucky
Francis Wayles Shine, New York, New York
John Lee Summerlin, Gainesville, Florida
Jesse L. Wagner, Reading, Pennsylvania
Nelson Shelly Weinberger, Sayre, Pennsylvania
William P. Wherry, Omaha, Nebraska
Casey Albert Wood, Pasadena, California
W. Stuart Woodruff, Colebrook, Connecticut

PRESIDENT LLOYD: It is very difficult at this time to say the appropriate thing with regard to Doctor Wherry, and I am reminded of what they said of the great architect in England: "If you would see his monument, you would look around you," and we can say the same thing of the Academy. More could be said, but I think you all realize at this point that is about all we can get through with.

We will call upon the Senior Member of the Council, Dr. Coates.

GEORGE M. COATES, M.D.: According to custom, it is the duty of the Senior Member of the Council to report to the members of the Academy at this time actions taken by the Council during this present meeting. The reports of the various committees of the Academy will be published in the TRANSACTIONS, so that only important details will be covered at this time.

The Council recommends:

1. That Dr. W. L. Benedict act on all committees to replace Dr. W. P. Wherry, deceased.

2. That Dr. Derrick Vail be sponsored by the Academy as its representative in England to observe methods of reconstructive surgery of the eyelids.

3. That a committee to be known as the Committee on Standardization of Tonometers be appointed as follows:

* Biographies have been published in the July-August 1942 TRANSACTIONS.

Dr. Francis H. Adler
Dr. Jonas S. Friedenwald
Dr. Adolph Posner
Dr. Sanford R. Gifford
Dr. Peter Kronfeld
Dr. Harry S. Gradle
Dr. D. O. Harrington

4. That the activities of the Diversion Club be extended to include entertainment features connected with the activities of the Academy.

5. The Executive Secretary announced that resignations have been received from the following 10 members, and they were accepted with regret:

Dr. Frederick L. Beck, Cheyenne, Wyoming
Dr. James G. Calhoun, St. Louis, Missouri
Dr. Frank E. Detling, Los Angeles, California
Dr. G. D. Hallett, New York, New York
Dr. A. E. Johnson, Watertown, South Dakota
Dr. Phillip Mysel, Boston, Massachusetts
Dr. Clarence Rainey, Wilmette, Illinois
Dr. D. L. Shumate, Kansas City, Missouri
Dr. Robert B. Sweet, Long Beach, California
Dr. Robert E. Wright, Dallas, Texas

6. Miss Clare McGovern was given by the Council the title of Registrar.

7. The advisability of incorporation of the Academy was discussed, and the Executive Secretary was authorized to obtain a legal opinion as to the advisability of this procedure.

8. Dr. Kaspar Pischel, of San Francisco, and Dr. E. C. Ellett, of Memphis, were recommended for honorary membership in the Academy.

9. It was ordered by the Council that those directing the Home Study Courses should proceed with the organization of clinical home study courses when and if conditions appear favorable.

10. The Council unanimously passed the following resolution:

"A memorial to Dr. W. P. Wherry is hereby established in the form of a Life membership in the Academy to be awarded to the outstanding candidate of the year as recommended by the American Board of Otolaryngology."

11. The Council recommends that Junior members in active military service pay one-half the regular dues for the calendar year 1943.

12. The Council recommends that the eligibility requirements for membership in the Academy be altered so as to permit the election to membership of maxillo-facial surgeons who have been certificated by the American Board of Plastic Surgery.

13. The Council recommends that Dr. Dean Lierle be appointed Chairman of the Committee on the Conservation of Hearing with power to reorganize it. It further recommends that one thousand dollars be appropriated for the use of the Committee for whatever purpose the Chairman may direct.

14. The Council recommends that the Committee on National Defense be discontinued. A new committee to be known as the Committee on War Activities was established. Dr. Burt Shurly was appointed Chairman with the power to appoint four additional members. The President and Executive Secretary are to be ex-officio members of this Committee.

15. The Council recommends that unless unforeseen developments occur, the next meeting of the Academy be held at the usual time in the Palmer House, in Chicago, and that the usual activities of the organization be carried on.

PRESIDENT LLOYD: Gentlemen, there are several important sections in the report and you may wish to discuss some of them before you act on it in toto. Suppose we go over each item. What is the recommendation?

HARRY S. GRADLE, M.D., Chicago, Ill.: Mr. Chairman, to bring it before the floor, I move that the report of the Senior Member of the Council be accepted.

ARTHUR J. BEDELL, M.D., Albany, N. Y.: I second the motion.

PRESIDENT LLOYD: It is regularly moved and seconded that the report of the Senior Member of the Council be accepted. Does anyone wish to discuss any detail thereof?

WALTER H. THEOBALD, M.D., Chicago, Ill.: I have no discussion except to commend the last item on the next meeting. I am commending the item which refers to the next meeting.

PRESIDENT LLOYD: Well, we have had excellent service here in Chicago and I think the Council has exercised very commendable wisdom myself. Is there any other detail that you wish to discuss? We are naturally anxious to save time, but not at the expense of getting the thing done in the right way.

Gentlemen, I take it that these matters, which have had the calm consideration of the Council, meet with your approval, so I will put the motion. All those in favor will say "aye"; all opposed. The motion is carried.

DR. BEDELL: Now, Mr. Chairman, I move you, sir, that the report be adopted. You have received it by that resolution.

DR. THEOBALD: I second the motion.

PRESIDENT LLOYD: Anything that will facilitate and come up to the legal requirements of the situation. Do you also wish to modify that motion to read "and that the officials are authorized and empowered and urged to carry out these recommendations in detail"? Do you want to add that?

DR. BEDELL: I do not think it is necessary.

PRESIDENT LLOYD: All right, the motion that Doctor Bedell makes is before you. All in favor say "aye"; all opposed. Now there is no question about this being done in the right way. When Doctor Bedell agrees on a thing it must be right.

Next are the Committee reports.

EXECUTIVE SECRETARY-TREASURER BENEDICT: Mr. Chairman and Members: The various Standing Committees have each given their annual report to the Council. These reports have been read and considered by the Activities Committee. There are no significant items that would require any change in our policies, and I should like to dispense with the reading of all these with your consent.

PRESIDENT LLOYD: They will be printed?

EXECUTIVE SECRETARY-TREASURER BENEDICT: They will be printed in the November-December issue of the TRANSACTIONS.

PRESIDENT LLOYD: Is the request of the Secretary agreed to? All in favor say "aye": all opposed. Carried.

Committee Reports

AMERICAN BOARD OF OPHTHALMOLOGY

OCTOBER 13, 1942

The Board has conducted seventy-six examinations to date and has issued 2,107 certificates.

Because many candidates for the Board certificate are in military service, the Board has suspended for the duration the requirements for case histories and written examinations. All other requirements, however, are retained and standards have not been lowered. Subjects previously covered in the written examination will now be included in the oral and clinical examination, which will require at least two days of examination of each candidate.

The Board also decided not to reduce the time requirements in the specialty, viz: three years in ophthalmology, and five years in otolaryngology combined with ophthalmology.

The Preparatory Group has been discontinued.

Because of the large number of candidates and the war emergency, the Board announces the following additional examinations:

New York City, December 13, 14, 15 and 16, 1942

Los Angeles, California, January 15 and 16, 1943

PERSONNEL OF THE BOARD

A.O.S.

Term ends 12-31-43—W. B. Lancaster
Term ends 12-31-44—John Green
Term ends 12-31-45—G. D. Theobald
Term ends 12-31-46—E. C. Ellett

Section on Ophth.

Term ends 12-31-43—Allen Greenwood
Term ends 12-31-44—S. Judd Beach
Term ends 12-31-45—Daniel B. Kirby
Term ends 12-31-46—Conrad Berens

Academy

Term ends 12-31-43—F. C. Cordes
Term ends 12-31-44—W. L. Benedict
Term ends 12-31-45—C. S. O'Brien
Term ends 12-31-46—A. B. Reese

REGISTRY OF OPHTHALMIC PATHOLOGY

July 1, 1941 - June 30, 1942

Advisory Committee

Dr. Harry S. Gradle, Chicago, Illinois, Chairman
Dr. F. H. Verhoeff, Boston, Massachusetts
Dr. Jonas S. Friedenwald, Baltimore, Maryland
Dr. Georgiana D. Theobald, Chicago, Illinois
Colonel J. E. Ash, Curator, Army Medical Museum (Ex-Off.)

TABLE I

YEAR	NO. OF ACCESSIONS
1922-1934	4,847
1935	415
1936	570
1937	863
1938	972
1939	1,167
1940	1,441
1941	1,353
	11,628

There were 1,353 accessions during the year, the decrease in the number from the preceding year being due to the fact that work on comparative anatomy has been discontinued during the war. An average of 112 enucleated eyes were received monthly from ophthalmologists in all parts of the country. There are now over 1,800 cases of malignant intra-ocular melanomas in the Registry, more than 600 of which have been followed 5 years or longer.

The course in ophthalmic pathology based on Registry material was given for the fifth year in connection with the Postgraduate Course in Ophthalmology at George Washington University.

The loan sets are still in demand by candidates for the Academy Board and by individual students. In addition, several students have spent some time at the Museum studying the slides.

An exhibit of comparative anatomy was prepared and shown at the Academy meeting last year and papers were read on malignant melanomas of the choroid and ciliary body and on epithelial tumors of the limbus. During the year a paper on equine periodic ophthalmia has also been prepared and published. It was read at the April meeting of the International Association of Medical Museums as a part of a symposium on comparative pathology.

The Atlas of Ophthalmic Pathology was revised and 1,000 copies were prepared. Through the courtesy of the Academy, complimentary copies were sent to the Surgeons General of the Army and Navy and to approximately 120 Army and Navy hospitals of their selection. Since the Atlases can be produced at a third of the original cost and in larger numbers, they have been adopted as texts in the Home Study Course.

There has been an appreciable increase in the number of cases received from Army personnel.

HARRY S. GRADLE, M.D., Chairman,
Advisory Committee,
Registry of Ophthalmic Pathology
J. E. ASH, Colonel, M.C., U. S. A.
Registrar

AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

September 1, 1942

UNFINISHED PROCEEDINGS

Optotypes

"Since the A.M.A. distance rating, which is made to conform to round percentages, is fairly satisfactory, it should be retained. The reading chart does not conform and should be revised to correspond with the distance charts. The committee wishes it understood that the charts are approved only for compensation purposes, not for scientific purposes, nor for recording vision of school children, factory employees, railroad employees or governmental services, including aviation, naval, military nor for any other purpose than that for which they were intended. The committee suggests that these instructions be printed on the charts. The committee stands ready to confer with the A.M.A. on this revision." (Signed) Alfred Cowan, sub-chairman.

Terminology in Motor Anomalies and Physiologic Optics

A preliminary draft has been prepared by sub-chairman, Lancaster.

Optometry

Some progress was reported. Final action was postponed indefinitely.

Visual Acuity Research

Progress is reported by sub-chairman Benedict.

New Instruments

Questions of the character of advertising of the biophotometer were referred to the Council on Physical Therapy of the American Medical Association. The Massachusetts Visual Test was also referred to the Council on Physical Therapy of the American Medical Association.

FINISHED PROCEEDINGS

Instruments

The committee favors the request of the Keystone View Company to exhibit at the Academy Meeting. Changes were made to meet former objections. Sub-committee reported to post office department its opinion concerning "spectro-chrome therapy" which was discussed in the American Medical Journal, November 9, 1941. The action was to debar agents representing such instruments from the use of the mails. A Test Chart submitted by Colonel Rowntree, M.C., U. S. A. was studied and criticized by the sub-committee, who reported to Colonel Rowntree and received his thanks.

New Appointments

1942-43. Charles A. Bahn, M.D.; Avery D. Prangen, M.D.; Alfred Cowan, M.D.

Ophthalmic Requirements of the Military Services

At the request of the committee, the visual requirements, qualifying and disqualifying conditions, and official sources of information in all branches of the Military Service were compiled by Dr. Bahn. This was published in the Archives of Ophthalmology, June, 1942.

CHARLES A. BAHN, M.D.

AMERICAN ORTHOPTIC COUNCIL

October 10, 1942

During the past year two noteworthy advances have been made.

1) Under the sponsorship of the American Orthoptic Council, an association of certified orthoptic technicians was established to cooperate with the Council in its educational activities.

2) The American College of Surgeons accepted the invitation to become a sponsor of the American Orthoptic Council along with the American Ophthalmological Society, the Section of Ophthalmology of the American Medical Association and the American Academy of Ophthalmology and Otolaryngology. Three of its fellows have been appointed to serve on the Council.

On September 17, 1941, a written examination was held throughout the country for 20 technicians, 3 of whom failed. The practical and oral examinations for the successful candidates were held in Chicago on October 17, 1941. Fifteen of the 16 candidates passed these examinations and were duly certified.

On June 5, 1942, an examination of technicians was held in New York City. Four technicians passed.

On August 29, 1942, a similar examination was held in San Francisco. Three technicians passed.

On October 1, 1942, a special examination was held in New York City. One technician passed.

There are now 66 certified technicians, 54 of whom are members of the American Association of Orthoptic Technicians.

Following the June examinations in New York City a meeting of the Council was held at which the By-Laws were revised to include the 3 delegates from the American College of Surgeons as Council members, and to include the President of the American Association of Orthoptic Technicians as an associate member ex-officio. It was also voted that the term of each member begin January 1st following his election.

A Committee on Standards has formulated definite standards for training of technicians and has approved certain instruments.

A successful symposium was held last year with the Association of Orthoptic Technicians. It is felt that such gatherings of technicians and Council members are most stimulating and should be encouraged.

The certificates issued to technicians continue to be the property of the American Orthoptic Council, returnable on demand and renewable annually at the discretion of the Council.

In addition to carefully examining candidates for orthoptic certificates, the Council proposes,

1) to continue its files on orthoptic conditions throughout the United States and Canada.

2) to enlarge its technicians directory so that it will contain complete information regarding every technician in the country.

3) to assist when requested in the formulation of seminars on orthoptics.

4) to assist reputable ophthalmologists in obtaining services of qualified technicians.

5) to cooperate with the Army, Navy and Civil Aeronautics Authority in working out plans whereby the services of qualified technicians can be used in the war effort at a minimum disturbance to civilian activities.

Well intentioned but misguided efforts toward including orthoptics in the Armed Services have already been noted and we deem it to be of the utmost importance that in the future orthoptic activities should be coordinated into a responsible organization representing American ophthalmology.

CHANGES IN PERSONNEL

At its June meeting the American Ophthalmological Society retired Doctor Edwin B. Dunphy from the Council and appointed to serve for 3 years Doctor LeGrand H. Hardy of New York City.

At its June meeting the Section on Ophthalmology of the American Medical Association retired Doctor George Guibor from the Council and appointed Doctor Walter B. Lancaster of Hanover, New Hampshire, to serve for 3 years.

Your Committee respectfully recommends:

1) That the American Academy of Ophthalmology and Otolaryngology continue its sponsorship and subsidization of this important work.

2) That it appoint for a 3 year period Doctor Avery Prangen of Rochester, Minnesota, to take the place of Doctor LeGrand H. Hardy, retiring.

LeGrand H. Hardy, M.D.
John Hitz, M.D.
Murray McCaslin, M.D.

COMMITTEE ON INDUSTRIAL OPHTHALMOLOGY

October 10, 1942

The members of the Committee on Industrial Ophthalmology, which is a joint committee made up of 3 members from the Academy and 3 from the section of the A.M.A., have been active during the past year. This committee has worked in conjunction with the Council on Public Health of the A.M.A.

The following is an outline of the present activities of the committee:

1. The compilation of a bibliography relating to many phases of the subject.
2. The investigation of visual requirements of many different types of employment in industry.
3. The study of available statistics relating to the percentage of employees with visual defects.
4. The analysis of data relating to the improvement in production (qualitative and quantitative) and brought about by correction of visual defects.
5. A study of the factors which may influence visual efficiency and seeing ability for specific types of jobs.
6. A survey of the prevalence of the practical known methods of preventing accidents.
7. A study of plant conditions with a number of medical staffs, personnel departments and management in order to learn first-hand visual problems of industry.

A number of papers have been read before various groups, both medical and lay, as a basis for debate and for obtaining the point of view of these interested groups.

A visual program for industry has been outlined and is being studied by members of the committee.

Many activities are still being conducted; therefore, the committee requests that it be continued.

John B. Hitz, M.D.

ADVISORY COMMITTEE TO EYE HEALTH COMMITTEE OF THE AMERICAN STUDENT HEALTH ASSOCIATION

June 1, 1942

The Advisory Committee has conferred with the Eye Health Committee in regard to progress of the examination of eyes of students in colleges of the United States that belong to the American Student Health Association. Under the auspices of the National Society for the Prevention of Blindness, an examination of the eyes of students entering two nursing schools in Minnesota have also been carried out and reported to the statistical division of the Society.

Since it was originally organized in 1936, the Eye Health Committee of the American Student Health Association has concentrated its efforts in the following main endeavors:

1. Improvement of college student lighting conditions.
2. Development of standard simple method of vision testing.
3. Development of the Luckiesh-Moss chart holder and illuminator.
4. The training of students in Teachers' Colleges in eye health.

The chart holder and illuminator is the result of painstaking study by the Advisory Committee of ophthalmologists and their cooperation with illumination engineers. This device now has the approval of the American Committee on Optics and Visual Physiology. The Welch Allyn Company of Auburn, New York, has accepted the responsibility of manufacturing these chart holders and has already sold about 100.

During the year the committee sent questionnaires to 216 of the leading colleges and universities of the country and although the response to these questionnaires was very poor, certain conclusions are warranted. There is not space to include here the total findings included in the responses received, but a brief summary of answers to 3 of the questions is recorded below.

Number of questionnaires mailed (September, 1941) 216
Number of colleges replying to questionnaire 53

Summary of replies:

1. Improvements in study surface and classroom illumination
   - None 19
   - Some 32
   - Not reported 2

2. Ophthalmological examination required
   - No 44
   - Yes 8
   - Not reported 1

3. Standard method of vision appraisal and Luckiesh-Moss Test Chart Illuminator used
   - No 40
   - Yes 12
   - Not reported 1

The National Society for the Prevention of Blindness handled the questionnaires and the tabulation of replies. Detailed analysis of this material is in the committee files. The National Society has from the start supported all the activities of the committee in a most generous way. Without the inspiration and material assistance of this organization the work of this committee would have been entirely impossible.

The returns from the questionnaire show that there is no lack of opportunity for state teachers' colleges interested in eye health of their students. It would seem that for the coming year emphasis should be laid on the suggestions already made by the Eye Health Committee and the Advisory Committee of ophthalmologists for:

1. More careful and comparable testing of the eyes of students.
2. More thorough medical examinations, including ophthalmological examinations. Retest vision annually.
3. More attention to correct lighting conditions for college classrooms, laboratories, libraries and student rooms.

The examinations for recruits show approximately 11 per cent of rejections because of eye defects. Since many of the students now in the colleges may be called within the near future, it becomes all the more essential to lay stress on the health aspects of college work. This rejection of 11 per cent would seem to indicate that somewhere along the line the school health program has been inadequate, either in its conception or in its development. Hence it seems all the more important that colleges prepare their students to understand as much as possible about eye health and the deviations from the normal.

The State of Washington has already formulated and is initiating a program for a course in eye health for the undergraduate teaching curriculum. This program includes lectures on the eye (anatomy, physiology, hygiene, refraction and refractive errors and common eye diseases), such lectures to be given by physicians specializing in eye diseases, lectures showing the relationship of the medical social worker in the eye health program, and such lectures to be given by medical social workers. In addition to the Washington program as drawn up, there should be included lectures on the relationship of the school health service to the eye health program, including demonstrations and practice in eye testing, such lectures and demonstrations to be given by the nursing service of the college; lectures showing the relationship of state and local departments of education to the eye health program, including the physical surroundings of the schools, the physical equipment, types of text books, maps, and so forth, and the necessity for special educational facilities for the education of partially seeing pupils, such lectures to be given by representatives of state and local departments of education and by the representatives of the department of education of the state teachers' college; lectures showing the relationship of state and local boards of health to the eye health of school children, such lectures not being confined to communicable diseases but rather to the whole health program as affecting the eye health program. Such lectures should be given by representatives of state and local boards of health.

Many cities and county health departments have on their staff a sanitary engineer whose services are available for lighting surveys and lectures on lighting and best practices in the use of equipment.

Eye Health Committee
LEE FERGUSON, M.D.
L. M. HICKERNELL, M.D.
J. H. KLER, M.D.
R. W. BRADSHAW, M.D., *Chairman*

Advisory Committee
LEGRAND HARDY, M.D.
HARRY S. GRADLE, M.D.
W. L. BENEDICT, M.D., *Chairman*

AMERICAN BOARD OF OTOLARYNGOLOGY

OCTOBER 13, 1942

Since 1924, when the Board of Otolaryngology was organized, 43 examinations have been given and 3,467 certificates issued.

Representing the Academy on the Board are T. E. Carmody, M.D., and Frank R. Spencer, M.D.

THOMAS E. CARMODY, M.D.

REGISTRY OF OTOLARYNGIC PATHOLOGY

JULY 1, 1941-JUNE 30, 1942

Advisory Committee

Dr. Joseph C. Beck, Chicago, Illinois, Honorary Chairman

Dr. Frank J. Novak, Chicago, Illinois, Chairman

Dr. Gregor W. McGregor, Toronto, Canada

Dr. Herman Z. Semenov, Los Angeles, California

Dr. Werner Mueller, Boston, Massachusetts

Dr. Bernard J. McMahon, St. Louis, Missouri

Colonel J. E. Ash, Curator, Army Medical Museum (Ex-Off.)

On June 30, 1942, there were 1,828 cases in the Registry, 222 having been added during the past year.

The loan sets have again been in great demand, particularly during the weeks preceding the Board examinations, when it is necessary to carry a waiting list. Eighty-eight otolaryngologists have used the sets since the first of the year. During the year the sets have been thoroughly revised and 5 new sets added. The syllabi have been completely redone.

Material was prepared and exhibited at the Academy meeting in October, 1941.

The Atlas of Otolaryngic Pathology was revised and 500 copies were prepared. Through the courtesy of the Academy complimentary copies were sent to the Surgeons General of the Army and Navy and to approximately 120 Army and Navy hospitals of their selection. Since it has been possible to produce the Atlases much cheaper and in larger numbers they have been adopted as texts in the Home Study Course.

Several temporal bones are in the process of preparation. Serial sections of temporal bones of a dog have been made for Dr. J. M. Robb, who is studying the effects of excess hormones on the inner ear, particularly as to the possibility of the production of otosclerosis. Serial sections were also made of pigeons' heads for Dr. Albert P. Seltzer, for study of the effect of occluding the external nares. The Medical Officers of the Army and civilian pathologists have been circularized in an effort to obtain material on which definitive studies can be made of the effects of aviation on the ear.

There has been an appreciable increase in the number of cases received from Army personnel.

FRANK J. NOVAK, M.D., *Chairman*
Advisory Committee, Registry of Otolaryngic Pathology
J. E. ASH, Colonel, M.C., U.S.A., *Registrar*

COMMITTEE ON CONSERVATION OF HEARING

OCTOBER 12, 1942

The Committee on the Conservation of Hearing at a special meeting held in the Palmer House on October 11, 1942, voted to submit to the Council the following motion:

It was voted to recommend that Dr. Horace Newhart be appointed Honorary Chairman of the committee and Dr. D. M. Lierle Executive Chairman, to reorganize the committee and appoint new members, subject to the approval of the President and Executive Secretary.

Dr. Ernest Seydell was appointed temporary secretary of this Committee.

HORACE NEWHART, M.D.

HOME STUDY COURSES

OCTOBER 1, 1942

During the first two years, the Home Study Courses were produced in a fairly regular pattern; but the advent of the War and the consequent reduction in the numbers of available faculty have necessitated a rather radical change in procedure. The greatest difficulty in the past has lain in the correction of the examination papers. That has been a real chore and all credit is due to the unselfish work of the faculty. These groups have performed this unsung labor of love without a whimper. Some of the registrants have complained about the delay in the return of their corrected examination papers, not realizing the enormous task involved; otherwise there have been but few complaints.

With the sharp reduction in the number of available faculty members, it was decided to limit the number of examination papers that would be corrected to 50 in Eye and 50 in E. N. T. Those registrants were to be bona fide Residents. All registrants over and above 50 in each specialty will receive the reading lists and the examination questions, but will not submit completed examination papers for correction. But they will receive a copy of a model examination paper, based upon the best answers among the 50 submitted for correction.

Each Group Leader in the faculty has been asked to select one of his men to correct the 50 examination pepers submitted. For this he is to be paid at the rate of 50 cents a paper. The corrections are to be made on the basis of constructive criticism. He will also be able to select the best answer to each question and the answers thus selected will be mimeographed into a model examination paper that will be sent to each registrant over the above 50 of each side.

As usual, there were many attempts to register after registration time was closed. In the case of Residents, it was felt that they should be accepted with limitations because they were still too young at the game to have gotten the information in proper time. But other graduate physicians have had ample notice through the columns of all the specialty journals and many of the general medical journals, noticeably the Journal of the American Medical Association. Consequently, many have been rejected because late registrations complicate the mechanics of the Courses to an almost insurmountable degree. The registrations for the year 1942-1943 are:

	Ophthalmology	*Otolaryngology*
For Correction	50	50
Not for Correction	113	28
Grand total		241

Numerous individual complications arise from time to time which your committee endeavors to adjust. Thus one man is ordered to foreign service and asks for a refund; another is returned to where he can complete his unfinished course of last year; a man in a small community cannot obtain the requisite books and has to be given a supplemental list, and so on. Many requests have been received for acknowledgement of the completion of the Course in order to obtain credit with the American Boards or the Armed Forces or Staff appointments. In accordance with directions from the Council, such requests are simply answered by a letter stating that "Dr. Blank has completed the Home Study Course in.....for the year 1941-1942."

Inasmuch as the finances of the Home Study Courses are handled through the central office, this committee is not in the position to submit financial details in this report. We can merely say that the Home Study Courses are self-supporting and are not costing the Academy anything.

Respectfully submitted,

H. S. Gradle, M.D.

SCIENTIFIC EXHIBIT

October 13, 1942

Because of the war, the number of exhibits has decreased.

Many exhibits had been scheduled to be given by younger men who were called into the Service later on. Thus their exhibits were cancelled.

There will be three awards, as previously, for ophthalmology and three for otolaryngology.

It is suggested that the scientific exhibits be limited to physicians and instructors and that commercial concerns not be permitted a place in the Scientific Exhibit.

O. E. Van Alyea, M.D.

COMMITTEE ON NATIONAL DEFENSE

October 13, 1942

In October, 1941, the Council of the Academy appointed a special committee for National Defense with the idea of cooperating with the Surgeons General in establishing a corps of efficient specialists in the Armed Services, and to aid in research problems developed by the emergency. Dr. Samuel Kopetzky, a member of the committee, conferred with the President of the United States and with the Surgeons General in order to determine their desires relative to the services of specialists.

The National Research Council appointed a Committee on Otolaryngology as a Sub-Committee of the Division of Surgery. Of this, Dr. Mosher was chairman. This subcommittee worked in conjunction with the committee of the A.M.A. in compiling the evaluation lists of otolaryngologists. It also met in Washington and made suggestions for the use of audiometers, the preservation of hearing, and discussed research problems.

The Army at first felt that the use of the audiometer was too laborious, but it is increasing its use. Your committee believes that accurate hearing records will be of great value in the establishment of a right to a pension after the war.

At a meeting of the Advisory Board for Medical Specialties held in Chicago, February last, Dr. W. P. Wherry and Dr. H. P. Mosher represented the American Board of Otolaryngology. Col. Geo. F. Lull of the Personnel Division of the Surgeon General's office, who attended by invitation, was asked if at that time the Surgeon General's office wanted specialists, and would specialists in the Army be assigned to their specialty? Col. Lull an-

swered that only young men for general use were then needed, and that they would be assigned to their specialty as soon as possible. Col. Lull added that specialists now serving in the Army who have not been certificated by the Examining Boards will be granted leave to take the examinations of the Boards when they can be spared.

The Sub-Committee on Otolaryngology cooperated with the Sub-Committee on Ophthalmology in publishing the second War Manual under the auspices of the National Research Council. The Surgeons General of the Army and the Navy said that the volume met their needs. The Otolaryngological Committee of the National Research Council deserves special commendation for the splendid work it has done in publishing this compact treatise on otolaryngology for use in the Army and Navy. Ophthalmology is covered in the first half of the book. The manual is valuable to those who require a concise statement of emergency problems as presented to the services.

Having finished its work the Committee on Otolaryngology was discharged. Otolaryngology, therefore, has no representative in the Medical Service of the Army or the Navy. There should be one. The National Research Council has retained its Sub-Committee on Ophthalmology.

A request for the appointment of consultants in corps areas, Navy centers, and the Surgeon General's departments was made, but as yet has not been acted upon favorably as far as Otolaryngology is concerned. However, the Surgeon General of the Army has recently appointed Col. Fred Rankin as consultant in surgery, Col. Morgan in medicine, and Col. Halloran in neuropsychiatry.

In the last war (1917-1918) hundreds of chronic running ears were found among enlisted men. This, of course, should not happen again, but it is. The Army feels that ear protectors are a bother, and too fussy to use. We feel that much preventive care is necessary.

The Surgeon General does not care for consultants, especially those around the age limits, in spite of physical fitness. Older men can release younger desk officers, and allow the latter to get out and fight. We believe this war will be won by fighting as in 1917-18, and this without continuous delay and change of plans. Physical disability of the minor sorts should be waived, and more medical officers would join the service. We do not believe that War Man Power Commissioner Paul V. McNutt is right in threatening medical men. In the last war, it was not necessary, and with proper assignments for specially trained men it will be voluntarily accomplished. The Surgeon General is finally using the evaluation of specialists as procured by the A.M.A. This fine job is now of great usefulness. The list of certificated members is in the hands of the Army and Navy as well as the names of new examinees as they passed. The confidential findings of not passed candidates are not turned in until they have had another chance at reexamination, as their records are not complete.

It is not surprising that the high ranking reserve officers are not assigned to active duty until the highly trained regular officers are promoted and placed in positions of command. The war needs are greater each month, and with ten million men in the service the reserve will be taxed beyond the capacity of the medical department to carry on. The record of the medical department under Generals Gorgas and Ireland stood every test in World War I, and it will again.

As the War goes on and the army increases in size, it is expected that our function as specialists will be more appreciated and valued. We are especially useful in reducing the number of pensions by the use of the audiometer, in the early treatment of infection and suppuration, and in preventive measures. We are useful also in preventing unnecessary operations and limiting radical surgery. It is rumored that, as in the last War, too much radical or "practice surgery" is being done by some of the younger men. The safeguard of hearing and infection are well in hand. Newer therapeutic discoveries and their early application will do much to curtail epidemics. The modern specialist is equipped and ready if his services will be given military recognition. At present the eye, ear, nose and throat specialist is in the hands of the consulting surgeon. It should be said, however, that some surgical chiefs of service do not interfere.

The cooperation of the otolaryngologists is ready and available at all times. We are anxious to be called on for any additional service that our Generals may desire. The sick and wounded and suffering will be cared for. Some of the consultants in France during the last war did not fit in well. The Regular Army medical men learned many bitter lessons at that time. Specialism is necessary now and it should be accorded its proper place in the armed forces. The eyes, ears and the respiratory organs of a great expanded army, navy, and air force will need the trained men of our specialty in greater numbers.

More postgraduate centers of instruction will be called for. There is a deficiency of special interns in many hospitals. This great Academy can help to train men for these positions and keep them coming.

BURT R. SHURLY, M.D., *Chairman*

EXECUTIVE SECRETARY-TREASURER BENEDICT: This is the report of the Honor Society, which met this noon. Elevation to the Honor Society is done by ballot. The result of the ballot is herewith given: Three members were elected to the Honor Society, Dr. James A. Babbitt, Philadelphia, Pennsylvania; Dr. Arthur J. Bedell, Albany, New York; and Dr. J. J. Shea, Memphis, Tennessee.

This requires no action here, Mr. President, as that Society is sufficient unto itself and they elect their own members.

PRESIDENT LLOYD: Life memberships.

EXECUTIVE SECRETARY-TREASURER BENEDICT: Those elevated to Life membership and Senior membership during the past year are:

Life Members

Anderson, W. B., Brownwood, Texas
Appleman, L. F., Philadelphia, Pennsylvania
Barber, F., Rochester, New York
Brust, H. O., Syracuse, New York
Colver, B. N., Los Angeles, California
Crisp, William Henry, Denver, Colorado
Davies, T. Alexander, Toronto, Canada
Duntley, G. S., Macomb, Illinois
Edwards, C. J., Vicksburg, Mississippi
Fawcett, I., Wheeling, West Virginia
Foster, J. H., Houston, Texas
Haskin, W. H., New York, New York
Henninger, L. L., Pasadena, California
Jacobs, S. M., New York, New York
Johnson, H. R., Fairmont, West Virginia
Jones, E. W., Lynn, Massachusetts
Krebs, Adolph, Pittsburgh, Pennsylvania
Lancaster, W. B., Hanover, New Hampshire
Lawrence, Granville A., Philadelphia, Pa.
Lischkoff, M. A., Pensacola, Florida
McGuire, H. H., Winchester, Virginia
McMurray, J. B., Washington, Pennsylvania
Monahan, J. A., Clinton, Massachusetts
O'Reilly, W. F., Lynn, Massachusetts
Porter, L. B., Providence, Rhode Island
Seydell, E. M., Wichita, Kansas
Shawhan, Glenn E., Nampa, Idaho
Sherman, Major A. Ed., Fort Rosecranz, Cal.
Tingley, Louisa Paine, Prividence, R. I.
Whisnant, A. M., Charlotte, North Carolina
Wurtz, W. J. M., Buffalo, New York

Senior Members

Atkinson, D. T., San Antonio, Texas
Beil, J. Wallace, Kansas City, Missouri
Beyer, Louis J., Buffalo, New York
Bordley, James, Jr., Baltimore, Maryland
Campbell, C. H., Columbus, Nebraska
Cary, G. C., Grand Junction, Colorado
Chase, E. Frank, Seattle, Washington
Curdy, R. J., Kansas City, Missouri
Decker, J. C., Sioux City, Iowa
Edwards, Slocumb, R., El Centro, California
Gale, M. Jean., Denver, Colorado
George, Henry W., Middletown, Pa.
Guyton, B. S., Oreford, Mississippi
Halsted, Fred S., Denver, Colorado
Henton, George E., Portland, Oregon
Herriman, L. L., Alamosa, California
Hodson, B. F., Miami, Florida
Irvine, A. Ray, Los Angeles, California
Johnston, Wilson, Portland, Oregon
Kress, George H., San Francisco, California
Lefler, Anna B., Los Angeles, California
Loomis, E. A., Minneapolis, Minnesota
McDiarmid, H. Oliver, Brandon, Man., Can.
Maiden, Sydner D., Council Bluffs, Iowa
Mitchell, E. K., Philadelphia, Pennsylvania
Morris, A. G., Rochester, New York
Neeper, Edward R., Colorado Springs, Colo.
Nisselson, Max, New York, New York
O'Connor, D. F., Newark, New Jersey
O'Connor, Roderic P., San Francisco, Calif.
Post, M. Hayward, St. Louis, Missouri
Ralston, William W., Houston, Texas
Reiche, O. C., Hazleton, Pennsylvania
Rickie, L. T., Trinidad, Colorado
Robertson, Edward N., Concordia, Kansas
Scarlett, Hunter W., Philadelphia, Pa.
Schiller, A. Noah, New York, New York
Silbernagel, C. E., Columbus, Ohio
Smith, D. S., La Crosse, Wisconsin
Spencer, W. H., Philadelphia, Pennsylvania
Sturges, Leigh F., New York, New York
Walker, C. E., Denver, Colorado

According to custom, buttons have been provided for Life members and Senior members. I am quite sure that a number of those whose names I have called are not here, but will those of you who are here whose names have been called please come to the desk and secure your buttons at the close of the meeting?

PRESIDENT LLOYD: The next order of business is the admission of candidates, and lists have been provided here of these names. We naturally do not care to read 207 names. They have been investigated and approved by the Council; so, gentlemen, the action on this list is now in your hands. What is your pleasure?

CARL H. MCCASKEY, M.D.: I move that they be accepted.

PRESIDENT LLOYD: Is it seconded?

LAWRENCE POST, M.D.: I second the motion.

PRESIDENT LLOYD: The motion is before you that membership be conferred upon the 207 candidates whose names appear on this printed list, which has the approval of the Council and recommendation by them that membership be conferred upon them. Is there any discussion? If not, all those in favor please say "aye"; all those opposed. It is carried.

The following candidates were elected to membership:

Elected to Membership

Abell, Harry Davis, Paducah, Kentucky, Op.
Alexander, Fred William, 10515 Carnegie Ave., Cleveland, Ohio, Oto.
Armentrout, Lewis Hamilton, Jr., Grace Hospital, Welch, W. Va., Oto.
Arnold, Ralph A., Capt. (M.C.), 65th Gen. Hosp., Ft. Bragg, N. D., Oto.
Austin, Marvin Fletcher, 30 N. Michigan Ave., Chicago, Ill., Oto.
Bacon, Lewis Heisler, 300 Mahantongo St., Pottsville, Pa., Op.
Baer, Alvin Julius (Major MC-AUS), Jefferson Barracks, Missouri, Op.
Baldwin, Gertrude Werder, 270 Commonwealth Ave., Boston, Mass., Op.
Baldwin, Stephen Glidden, Adams Building, Danville, Ill., Oto.
Barnes, Mark Hopkins, 324 E 41 St., New York, N. Y., Op., Oto.
Barone, Michael Horace, 468 Delaware Ave., Buffalo, N. Y., Oto.
Barry, William B., Univ. of Kansas Hospitals, Kansas City, Kan., Oto.
Benkwith, Karl Burton, Bartlett Bldg., Montgomery, Ala., Op.
Bertrand, George Joseph, 93 College St., Montpelier, Vt., Op.
pelier, Vt., Op.
Blankstein, Samuel S., 606 Wisconsin Ave., Milwaukee, Wis., Op.
Boden, Worthey Carl (Capt. M.C.), Davenport Bk., Bldg., Davenport, Iowa, Oto.
Bonner, William Richard, 3 W. White St., Summit Hill, Pa., Op.
Bornstein, Max, Heyburn Bldg., Louisville, Kentucky, Oto.
Bradish, Robert Francis (Major M.C.), New Orleans Port of Embarkation, Poland and Dauphin St., New Orleans, La., Op.
Bridgett, Frank Aloysius, 3330 Chestnut St., Philadelphia, Pa., Oto.
Brown, Andrew George, Du Pont Bldg., Miami, Florida, Op.
Bryan, William T. K., 3720 Washington Blvd., St. Louis, Mo., Oto.
Butler, Edmund John, 25 Garden St., Cambridge, Mass., Oto.
Capeles, Thomas Francis, 191 Merrimack St., Haverhill, Mass., Oto.
Chandler, Arthur Cecil, Medical Arts Bldg., Charleston, W. Va., Op.
Chenoweth, Charlie Everette, Forester's Bldg., Mason City, Ia., Oto.
Clayton, Milton Burns, Statesville, North Carolina, Oto.
Cohen, Jerome Harry, Johnstown Bank & Trust Bldg., Johnstown, Pa., Op.
Collins, Braswell Elisha (Capt. M.C.), Station Hospital, Army Air Base Hunter Field, Savannah, Georgia, Oto.
Combs, Arnold Brefoe, 200 West Second St., Lexington, Kentucky, Oto.
Combs, Fielding, Nissen Bldg., Winston Salem, North Carolina, Oto.
Connelly, Edgar James, 7 Roshek, Bldg., Dubuque, Iowa, Oto.
Connor, Arthur B., Sweet Bldg., Ft. Lauderdale, Florida, Oto.
Connor, Joseph Joyce, 750 Main St., Hartford, Connecticut, Op.
Creveling, Earle LaMont, 17 N. Virginia St., Reno, Nevada, Oto.
Cunningham, Henry Kelso, Medical Arts Bldg., Knoxville, Tennessee, Oto.
Cutler, Harold Masha, 50 Shirley St., Old Town, Maine, Oto.
Dame, Lawrence R., 57 Federal St., Greenfield, Mass., Op.
Davis, Leo Lloyd, 1437 Monaco Parkway, Denver, Colorado, Op.
Decker, Phillip Harold, 25 W. Third St., Williamsport, Pa., Oto.
Deutsch, William, Jr., (Capt. M.C.), Station Hospital, Camp Barkeley, Texas, Oto.
Diess, Robert Graham, 121 S. Highland Ave., Pittsburgh, Pa., Op.
Dix, Harold C., 405 N. Charles St., Baltimore, Maryland, Op.
Duboe, Sidney Harvey (Capt. M.C.), Camp Barkeley, Texas, Oto.
Dunnavan, Floyd Leighton, Arts Bldg., Vancouver, Washington, Op.
Edelman, Maurice Irwin, 30 N. Michigan, Ave., Chicago, Ill., Oto.
Edwards, Gwilym Austin, Home Guards Bldg., Van Wert, Ohio, Oto.
Ehrmann, Evelina W., 327 So. LaGrange Road, LaGrange, Ill., Op.

Elfman, Louis Kenneth, 6330 Lebanon Ave., Philadelphia, Pa., Oto.
Ernsting, Harry Carl, 8 West Main St., Springfield, Ohio, Op.
Falkson, Kurt F., 229 W 78th St., New York, N. Y., Oto.
Farrior, J. Brown, Univ. Hospital, Ann Arbor, Michigan, Oto.
Field, Homer Baker, 2433 W. Prairie, Blue Island, Ill., Op.
Fisher, Gilbert Eugene, Medical Arts Bldg., Birmingham, Ala., Oto.
Fisher, Guy Rothwell, One E Beverley St., Staunton, Virginia, Oto.
Flanders, Merton Newcomb, Tilton General Hosp., Ft. Dix, N. J., Oto.
Ford, Leroy Sargent, 30 School St., Keene, New Hampshire, Oto.
Foster, Clarence Batchelder (1st Lt. M.C.), Wm. Beaumont General Hospital, El Paso, Texas, Op.
Gadomski, Casimir Francis, 331 So Broad St., Elizabeth, N. J., Op.
Gallizzi, Vincent D., 51 So. Main St., Pittston, Pa., Oto.
Gambill, Perry Judson, Greystone Park, N. J., Oto.
Geary, Russell Diener, 337 Bridgeboro Road, Riverside, N. J., Oto.
Geeseman, Gorge Robert, 104 W Pike St., Canonsburg, Pa., Op.
Gidoll, Sidney H., 516 Sutter St., San Francisco, Calif., Oto.
Goetzman, Arthur Cecil, 8907 Hennepin Ave., Niagara Falls, N. Y., Op.
Goldberg, Herman Krieger, 8 East Eager St., Baltimore, Md., Op.
Goodhill, Victor, 1101 Wilshire Medical Bldg., 1930 Wilshire Blvd., Los Angeles, Calif., Oto.
Gorin, Meyer, 35 Chestnut St., Rochester, New York, Oto.
Gorrilla, Laurence Vincent, 3115 South Grand Blvd., St. Louis, Mo., Op.
Gowens, Henry Lytle Jr., 123 So 23rd St., Philadelphia, Pa., Op.
Grove, Bruce Alexander, 141 E Market St., York, Pa., Op.
Gundrum, Lawrence Kramer, 1930 Wilshire Blvd., Los Angeles, Calif., Oto.
Haas, Louis Richard, Smith Clinic, Pittsburg, Kansas, Op., Oto.
Hagan, Cornelius Edward, Jr., 151 Rock St., Fall River, Mass., Oto.
Hageman, George Ricker, Longmont Hospital, Longmont, Colorado, Oto.
Hall, Colby, 1136 W 6th St., Los Angeles, California, Oto.
Hallberg, Olav Erik, Mayo Clinic, Rochester, Minn., Oto.
Hamilton, Howard Eugene, 10515 Carnegie Ave., Cleveland, Ohio, Oto.
Hanckel, Richard White, Jr., 96½ Bull St., Charleston, S. C., Oto.
Hannon, Robert Emmet (Capt. M.C.), U. S. A., Okla. City Air Depot, Oklahoma City, Oklahoma, Oto.
Harkins, Herbert Perrin, Merion and Montgomery Aves., Bryn Mawr, Pennsylvania, Oto.
Harrington, Silas Frederick, Medical Arts Bldg., Dallas, Texas, Oto.
Harris, Harold Earl, Cleveland Clinic, Cleveland, Ohio, Oto.
Hayden, Jerome William, 4458 Madison St., Chicago, Ill., Oto.
Henry, Robert, 105 So Mill St., Colfax, Washington, Op.
Hicks, William Wynne, 200 E 6th St., Ellensburg, Washington, Oto.
Hogsett, Smith Fuller, Jr., 4618 Chester Ave., Philadelphia, Pa., Op.
Holstein, Theodore, 105 N 8th St., Boise, Idaho, Op.
Houser, Ben Philip, 214 W. Broad St., Tamaqua, Pa.
Howard, John Clair, Professional Bldg., Kansas City, Missouri, Oto.
Howell, Homer Preston (Capt.), Hofmann Bldg., Ottumwa, Iowa, Op.
Hubble, William Fields, Citizens Building, Decatur, Ill., Oto.
Hutchison, Lloyd Snyder, 233 N Duke St., Lancaster, Pa., Oto.
Insel, Herman Herbert, Welfare Island, Metropolitan, New York, N. Y., Oto.
Irwin, John Joseph, Baronne Building, New Orleans, La., Oto.
Irwin, Thomas M. (Capt. M.C.), Station Hospital, Camp Stewart, Ga., Oto.
Irwin, Willard James, Medical Arts Bldg., Knoxville, Tennessee, Oto.
Jaffe, Morris, 940 Grand Concourse, New York, N. Y., Op.
Jessel, Erwin, 1749 Grand Concourse, Bronx, New York, N. Y., Op.
Johnston, William Raby, 1515 State St., Santa Barbara, Calif., Oto.
Kaplan, Leon, 881 Lafayette St., Bridgeport, Conn., Op.
Katz, Harry Kolman, 1206 Cottman St., Philadelphia, Pa., Op.
Kenyon, Byron, Benjamin, Bronxville Trust Co. Bldg., Bronxville, New York, Oto.
Kerst, John Arthur, R. R. No. 4, Springfield, Ill., Op.
Kopp, Mortimer M., 1300 Church Ave., Brooklyn, N. Y., Oto.
Krall, J. Thomas, 1927 Spruce St., Philadelphia, Pa., Op.

Krone, William Frank, 31 Lincoln Park, Newark, N. J., Oto.

Lamberson, Frank Abrom, 19600 Grand River, Detroit, Michigan, Oto.

Lathbury, Edwin F., 333 Linwood Ave., Buffalo, N. Y., Oto.

Lewin, Michael L., 169 E 78 St., New York, N. Y., Oto.

Lewy, Alfred, 25 E Washington St., Chicago, Ill., Oto.

Lindberg, Vernon Leslie, Physicians and Surgeons' Bldg., Minneapolis, Minnesota, Op.

Lockwood, Chester Clinton, 4707 Broadway, Chicago, Ill., Op.

Loeb, William Joseph, Station Hospital, Ft. Thomas, Kentucky, Oto.

Lynch, Joseph Sidney, 525 Welsh St., Chester, Pa., Op.

McAlpine, Paul Theodore, 129 Summit Ave., Summit, N. J., Op.

McBride, Linn Frederick, 122 S. Michigan Ave., Chicago, Ill., Oto.

McCollom, Robert Lucien (Major M.C.), Dow Field, Bangor, Maine, Oto.

McCullough, Lester E., 1553 Woodward Ave., Detroit, Michigan, Op.

McGehee, Melvin Wood, Washington & 6th Ave., Montgomery, W. Va., Oto.

McLane, Jesse Newman, Blount Bldg., Pensacola, Fla., Oto.

Marks, Moses Isaac, 10515 Carnegie Ave., Cleveland, Ohio, Oto.

Marks, Paul Lombroso (1st Lt. M.C.), Station Hospital, Camp Livingston, Louisiana, Op.

Mather, Homer Raymond, First Nat'l Bank Bldg., Latrobe, Pa., Oto.

Mattis, Robert Dean, 634 North Grand Ave., St. Louis, Mo., Op.

Mensher, Ira Wadsworth, 50 Plaza St., Brookyln, N. Y., Op.

Meyers, Paul Thomas, U. S. Bank Bldg., Johnstown, Pa., Oto.

Miller, Edwin Ephron, Medical Arts Bldg., Knoxville, Tenn., Oto.

Moffat, Harold Forster, 881 Young St., Honolulu, Hawaii, Op.

Morgan, Harold Eugene, 210 South Pine St., Newton, Kansas, Op.

Mosher, Henry Adams, Reibold Bldg., Dayton, Ohio, Op.

Mountain, Edward Russell, 138 W 9th St., Erie, Pa., Oto.

Neidlinger, William J., 576 Farmington Ave., Hartford, Conn., Oto.

Neuberger, Julius Frederick (Comdr. M.C. U.S.N.) U. S. Naval Hosp., Brooklyn, N. Y., Oto.

Nodine, Edwin Rogers, Commercial Bank Bldg., Andalusia, Ala., Oto.

Nugent, Maurice Wilfrid, Port Colborne, Ontario, Canada, Op.

O'Connor, Thomas Philip, 30 N. Michigan Ave., Chicago, Ill., Oto.

Parker, Ray, U. S. Bank Bldg., Johnstown, Pa., Oto.

Pastore, Peter Nicholas, Medical College of Virginia Hospital, Richmond, Virginia, Oto.

Peden, Barton Edward, Stimson Building, Seattle, Washington, Oto.

Pegues, James Cary, 301 Howard Street, Greenwood, Miss., Oto.

Pellettiere, Edmund Victor, 1530 N. Damen Ave., Chicago, Ill., Oto.

Petruzzi, William Astor, 65 Pondfield Road, Bronxville, N. Y., Oto.

Phelan, Harold V., Rose Bldg., Cleveland, Ohio, Op.

Phillips, James Winston, Hurst E.E.N.T. Hospital-Clinic, Longview, Texas, Oto.

Potter, William Bently, University of Texas Medical College, Galveston, Texas, Op.

Priest, Robert Edward, 4344 Grand Ave., Western Springs, Ill.

Pumphrey, Gordon Hughes, 100 North Main St., Mount Vernon, Ohio, Op.

Rosedale, Raymond Samuel, 610 N. Market Ave., Canton, Ohio, Oto.

Ramsay, Robert Matthews, 320 Yale Ave., Winnipeg, Canada, Op.

Reed, Elmer McCready, 1419 Seventh Ave., Bever Falls, Pa., Oto.

Reeder, James Ellery Jr., 505 6th St., Sioux City, Iowa, Op.

Richardson, George A., Northwestern Medical School, Room 279, Chicago, Illinois, Oto.

Riggs, Ralph Hearsey, Medical Arts Bldg., Shreveport, La., Oto.

Rizzuti, A. Benedict, 160 Henry St., Brooklyn, N. Y., Op.

Roberts, Gilbert Jay, 586 North Main St., Pomona, Calif., Oto.

Rohm, Robert Franklin, 7026 Jenkins Arcade, Pittsburgh, Pa., Op.

Roper, Kenneth Lawrence, Clinical Division, Dartmouth Eye Institute, Hanover, N. H., Op.

Rudolph, Edward, Aberdeen, South Dakota, Op.

Russell, Martin V., 200 W Elm St., El Dorado, Arkansas, Oto.

Russell, Sidney Benteen, Midelfart Clinic, Eau Claire, Wisconsin, Op.

Sarno, J. Frederick, 109 So. William Street, Johnstown, New York, Oto.

Saskin, Edward, 855 St. Marks Ave., Brooklyn, N. Y., Op.

Scanlon, Wilko Gruver, 201 N. El Molino Ave., Pasadena, Calif., Oto.

Schoolman, Joseph G., Stark General Hospital, Charleston, S. C., Oto.

Schwartz, Harry, 1749 Grand Concourse, New York, N. Y., Op.

Seltzer, Albert Pincus, 1332 N. Franklin St., Philadelphia, Pa., Oto.

Shilling, Raymond LeRoy, 408 Center St., Ashland, Ohio, Oto.

Siewert, Otto Lucht, 408 North St., Logansport, Indiana, Op.

Simons, Leander T., Station Hospital, Ft. Leonard Wood, Mo., Op.

Sims, J. Lawrence, Station Hospital, Selfridge Field, Michigan, Oto.

Smart, Robert Edwin, Medical Arts Bldg., Ottawa, Ontario, Canada, Op.

Smith, Herschel Stanley, MRTC, Camp Grant, Ill., Op.

Smith, Rodger Browning, 2065 Adelbert Road, Cleveland, Ohio, Oto.

Snitman, Maurice F., 55 E Washington St., Chicago, Ill., Oto.

Spearman, Maurice P. S., Box 751, Kingsville, Texas, Oto.

Stewart, James V. (Lt. M.C.), Station Hospital, Cochran Field, Macon, Georgia, Oto.

Stockwell, Eunice LeBaron, 2018 Pine St., Philadelphia, Pa., Op.

Stoner, Cyrus Hubert (Major M.C.), Dale Mabry Field, Tallahassee, Florida, Op.

Strong, James Craig, Jr., 5 W. Fourth St., Cincinnati, Ohio, Op.

Struble, Gilbert Carl (Major M.C.), Billings General Hospital, Ft. Benjamin Harrison, Ind., Op.

Sullivan, Tim, Central Trust Bldg., Sterling, Ill., Oto.

Sykes, Edwin Meredith, Nix Bldg., San Antonio, Texas, Oto.

Terrell, Vance, Stephenville, Texas, Oto.

Thompson, James Parvin, Miller Bldg., Yakima, Washington, Op.

Thompson, Lester, Emmit, Woolworth Bldg., Salida, Colorado, Oto.

Thornburgh, Robert Grant, Professional Bldg., Long Beach, Cal., Op.

Tomlinson, John Wesley, 900 Chester Pike, Sharon Hill, Pa., Op.

Trainor, George McCague, 277 Alexander St., Rochester, New York, Oto.

Trott, Leslie De Nyse, 143 N Brand, Glendale, California, Oto.

Trueman, Robert Heiserman, Stark General Hospital, Charleston, S. C., Op.

Tucker, Frank Landale, Jr., 464 9th St., Brooklyn, N. Y., Oto.

Walker, Oliver Frank, 2873 Lewiston Road, Niagara Falls, N. Y., Oto.

Wall, John William, 1180 E 63rd St., Chicago, Ill., Oto.

Waltz, Harold Darnell, (Major M.C.), Ellington Field, Houston, Texas, Oto.

Washick, Frank, 501 Cotman Ave., Philadelphia, Pa., Oto.

Weinberg, Herman Charles, 10515 Carnegie Ave., Cleveland, Ohio, Op.

Weisser, Charles William, May Building, Pittsburgh, Pa., Op.

White, Carl Harper, 714 Beach St., Flint, Michigan, Oto.

White, Clarence Hunt, Maria Parham Hospital, Henderson, N. C., Oto.

Wieselthier, Maurice L., 1322 Union St., Brooklyn, N. Y., Op.

Wilkins, Ellis LeRoy, 266 Yazoo (McWilliams Bldg.,) Clarksdale, Miss., Oto.

Wilson, Otis M., 502 Third St., Wausau, Wisconsin, Op.

Wilson, William Hildebrand, Mayo Foundation, Rochester, Minn., Oto.

Wishart, David Edmund Staunton, Medical Arts Bldg., Toronto, Ontario, Canada, Oto.

Woolsey, William Jenkins, 212 N. 7th St., Waco, Texas, Op.

Zbar, Joseph Edward, 411 Hempstead Ave., Rockville Centre, N. Y., Oto.

PRESIDENT LLOYD: The next item is one of some importance. We will call upon Doctor Coates for the report on the nomination of officers.

DR. COATES: The Council takes pleasure in recommending the nomination of the following officers for the ensuing year:

President — James A. Babbitt, M.D., Philadelphia

President-Elect — Lawrence T. Post, M.D., St. Louis, Mo.

First Vice-President — Sanford R. Gifford, M.D., Chicago

Second Vice-President — Wesley Bowers, M.D., New York, N. Y.

Third Vice-President — William Krieger, M.D., Poughkeepsie, N. Y.

Comptroller — Secord H. Large, M.D., Cleveland, Ohio

Executive Secretary-Treasurer — Wm. L. Benedict, M.D., Rochester, Minn.

Secretary, Ophthalmology — Algernon B. Reese, M.D., New York, N. Y.

Secretary, Otolaryngology — O. E. Van Alyea, M.D., Chicago, Ill.

Secretary, Instruction, Ophthalmology—A. D. Ruedemann, M.D., Cleveland, Ohio
Secretary, Instruction, Otolaryngology—Dean M. Lierle, M.D., Iowa City, Iowa
Secretary, Public Relations—Erling W. Hansen, M.D., Minneapolis, Minn.
Member of Council (Otolaryngology)—Carl H. McCaskey, M.D., Indianapolis, Ind.
Liaison Officer, Honor Society—E. C. Ellett, M.D., Memphis, Tenn.
American Board of Ophthalmology—Algernon B. Reese, M.D., New York, N. Y.
American Board of Otolaryngology—Frank R. Spencer, M.D., Boulder, Colo.
American Association for the Advancement of Science—Wm. L. Benedict, M.D., Rochester, Minn.
American Committee on Optics and Visual Physiology—A. D. Prangen, M.D., Rochester, Minn.
American Orthoptic Council—A. D. Prangen, M.D., Rochester, Minn.
Eye Health Committee of the Student Health Association—Lawrence T. Post, M.D., St. Louis, Mo.

PRESIDENT LLOYD: Gentlemen, what is your pleasure with this list?

WILLIAM HENRY CRISP, M.D.: I move that the report be approved, the nominations closed and the Secretary be instructed to cast the ballot accordingly.

DR. THEOBALD: I second that motion.

PRESIDENT LLOYD: It is regularly moved and seconded that these officers as named here be elected by the Secretary casting a single vote indicating the approval of this Society. All those in favor will say "aye," please. Are there any on the other side of the fence? There seem to be none. Mr. Secretary, will you cast one vote simultaneously for all these nominees?

EXECUTIVE SECRETARY-TREASURER BENEDICT: I vote "yes."

PRESIDENT LLOYD: It is so ordered.

(Doctor Babbitt was called to the chair.)

DR. BABBITT: Fellows of the Academy: In very real friendship, conversation plays a very little part. Hours go by and in the company of a friend it is not necessary to speak. Words will feebly express the gratitude and appreciation of your confidence in nominating and electing me to this position. One undertakes it, having known the work of this Academy and having seen the immense scope of it in these days of the Council session. One hesitates much in undertaking such a job. Our genial President, as I have watched him this year, the happy approach to everybody, the quick wit and quick decisions, his keen mind on the essentials of the work, has given a sort of pattern which might be followed. The untiring work of the Board of Secretaries leaves little for the President to do, but there are things in this war year that are going to cause some anxiety. However, those problems we will not undertake until they come before us.

All I want to do now is to thank you over and over again for your confidence in me in electing me.

PRESIDENT LLOYD: Now it is a great privilege to introduce Doctor Post. We want to acknowledge our pleasure at having you with us, and I am sure the gentlemen would like to hear a word from you, too.

DR. POST: Mr. President and Fellow Members: I cannot adequately express my great appreciation for election to this high office. The Academy has always been to me the ideal Society. We perhaps have a few dark years ahead of us. We do not know what the future holds for any of us as individuals nor what it holds for the Academy, that is, the immediate future, but we do know that the ultimate future is bright and that the Academy will come through even better than ever. I can only say that I hope to serve you to the best of my ability. Thank you.

PRESIDENT LLOYD: Now we come to the end of the program.

DR. BEDELL: Mr. Chairman, before the meeting is adjourned, I move you, sir, that the newly elected President, Doctor Babbitt, take the chair and receive a resolution of thanks to our retiring President and to our newly-elected manager and all who will serve with them.

The motion was seconded.

Doctor Babbitt assumed the Chair.

CHAIRMAN BABBITT: I will inform the gentleman that the new President does not actually take office until January, but he will entertain this resolution with great pleasure.

You have heard this resolution. It has been seconded. I will ask for a rising vote.

Those present arose and applauded.

The meeting adjourned at six o'clock.

Society Proceedings

AMERICAN MEDICAL ASSOCIAION
SECTION ON OPHTHALMOLOGY
June 10, 11 and 12, 1942
Atlantic City
LAWRENCE T. POST, M.D., Chairman
DERRICK VAIL, M.D., Secretary
Ninety-Third Annual Session

Chairman's Address: Lifelong Care of the Eyes. Lawrence T. Post, St. Louis

The Effects, Other Than Anti-Infectious, of the Sulfonamides on the Eye. Moacyr E. Alvaro, Sao Paulo, Brazil

Different effects of sulfonamides on the eyes of man according to data found in the literature. Different interpretations as to the pathogeny of the different types of changes. The usual therapeutic anti-infectious effects are not mentioned. A series of patients to be submitted to sulfonamide treatment was examined before the treatment as to visual acuity, muscle balance, accommodation, light perception, color perception, ocular tension and pressure of the central artery of the retina, results of these examinations being then compared with findings during the treatment.

SYMPOSIUM ON GERIATRICS

Urologic Geriatrics. John H. Morrissey, New York

Among conditions frequent in elderly men are changes of the structures in and about the bladder neck. A fair proportion of these show malignant changes. These seem to be accompanied rarely by associated changes in the eye and adnexa. There is a sufficient proportion in which eye disease is present, either in association, or as a complication, so that both the ophthalmologist and the urologist should bear in mind the possibility of one condition complicating the other and be familiar with the initial symptoms. Relative degree of occurrence of these complications in different clinics; end results. Recent changes in the operative procedures on the prostate, both in benign and in malignant types of obstruction, are less debilitating to the patient. These are discussed from the point of view of the lessened incidence of ophthalmologic complications. Endocrine therapy of certain types of senile atrophic changes in the female genitalia. Report of results.

Medical Geriatrics (Lantern Demonstration). George M. Piersol, Philadelphia

Geriatrics is assuming added importance as longevity increases. We are rapidly becoming a nation of older people. This situation is fraught with significance, not only medical but also of a socio-economic-political nature. The increase in life expectancy is due largely to control of conditions destructive of youth. Preventive medicine by saving life among the young has brought about a significant increase in the number of old people. They live to be the victims of degenerative diseases. This has an effect on medical practice. To meet these demands of our older population we must acquire a better understanding of the physiologic, pathologic and medical problems of old age. The conditions most conspicuous in older people are diseases of the cardiovascular-renal apparatus, metabolic disorders, neoplastic disease and nutritional disturbances. Many of these conditions present complications of interest to the ophthalmologist; in others their early diagnosis depends on his observations.

Neuropsychiatric Geriatrics (Lantern Demonstration). Henry W. Woltman, Rochester, Minn.

Aging itself is not a disease but a normal evolution that in some tissues begins in youth and in all tissues takes place eventually. It varies in tempo as to person and organ, some of which, because of their importance, appear to be affected in particular. Both form and function of the nervous system take part in this process with more or less striking results. In old age certain complaints and diseases occur with obvious frequency, partly because the persons concerned have been spared until at last they fall victims, partly because the aging organism furnishes certain requisites for their development. Some disorders peculiar to no age are altered in their course and behavior when they occur later in life. As during youth and middle age, so during the senium, it

is wise and charitable to consider the patient as well as the disease.

Ophthalmic Geriatrics (Lantern Demonstration). Conrad Berens, New York

Life expectancy fifty years ago was 45 years for white people; today it is between 60 and 65 years. Since 1930 the number of people in the 46 to 64 year classification has increased 2.3 per cent, and those in the 65 year and over group, 1.4 per cent. Because our practice will be increasingly concerned with eye conditions observed in older people, it seems timely to consider some of the more striking changes in the eye and adnexa in the aging process. Pathology and functional changes. Among the changes described are ptosis, remote near point of convergence, pinguecula, thickening of Tenon's capsule, fatty degeneration of the tunica fibrosa, arcus senilis, shallowing of the anterior chamber, rigidity of the iris, lessening of the function of the ciliary muscle, irregularity of the capillary network of the choroid, drusen, senile cataract, presbyopia, retinal degenerations and retinitis due to toxic agents.

Fibroblastic Overgrowth of Persistent Tunica Vasculosa Lentis in Infants Born Prematurely (Lantern and Motion Picture Demonstration). T. L. Terry, Boston

A bilateral fibroblastic overgrowth behind the crystalline lens appears to be a new disease entity occurring in a small percentage of infants born prematurely. One necessary etiologic factor must lie in extreme premature birth because none of the infants with this condition weighed more than 2,000 Gm. (4 pounds) at birth, a stage at which the hyaloid artery is still functioning, and because the anomaly was not observed in any of the infants before 4 months of age. The infrequency with which this condition is observed in infants born extremely prematurely indicates that at least one other etiologic factor must be present. List of factors that may be included. Experimental work on various pregnant animals and their young, such as opossums, rats, pigs and cats, to determine the possible importance of these factors.

The Nature, Scope and Significance of Aniseikonia (Lantern Demonstration). Walter B. Lancaster, Hanover, N. H.

Sensations from the two eyes are not identical and are not intended to be, in the best of eyes. Many of the differences or discrepancies are essential, since they supply the clues that tell us where things are (space sense), often more important than telling what they are (form sense). Vision is not a simple affair. There is no such thing as a simple isolated sensation. No one has a grasp of the vast subject of how we see who does not comprehend the role played by the differences in the dioptric and in the perceptual images of the two eyes, since on these are based the refinements of space perception. In some cases these differences (for which we coined the name aniseikonia) exceed useful limits and give rise to eyestrain of the type familiar to most ophthalmologists. This is only one phase of the great subject of aniseikonia.

Discussion

Dr. Ernest A. W. Sheppard, Washington, D. C.: Dr. Lancaster has defined, classified and presented the subject logically, simply and briefly.

The following classification has helped me to visualize this difficult and involved subject:

1. Physiologic: That which Dr. Lancaster has described as normal.

2. Inherent: That which exists with emmetropia or isometropia. This type might be considered as anatomic, essential or inherent.

3. Induced: (a) that induced by the correction of anisometropia or antimetropia; (b) that induced by changes in base curve or thickness or by distance of the lens from the eye.

The importance of the induced type in everyday refraction is demonstrated by the following cases:

Case 1. — J. R. P., a man aged 65, was examined because of presbyopic symptoms. He was emmetropic and needed a + 2.50 D. sphere for reading. The ocular examination otherwise gave essentially normal results. His prescription was ground in ultex A and neutralized correctly. He complained that while wearing his glasses he would drive his car too far to the left. The right lens was ground on a —8.00 D. base curve and the left on a —6.00 D. base curve. This difference in the base curves introduced aniseikonia or added to inherent aniseikonia sufficiently to disturb his perception of spatial relationships.

Case 2. — C. H., a man aged 73, for whom a cataract in each eye had been successfully removed, saw clearly with each eye but complained that objects seen with the left eye were larger and that binocular vision was not comfortable. The findings were as follows:

Right eye: + 6.50 D. sph. ⊃ + 1.75 D. cyl., axis 165, vision 20/20 —4; left eye: + 8.00 D. sph. ⊃ + 1.00 D. cyl., axis 45, vision 20/20 —1.

Right eye: 9.75 mm. from the closed lid; left eye: 11.50 mm. from the closed lid.

Right eye: front curves, + 10.50 D. and + 12.25 D.; left eye: front curves, + 12.00 D. and + 13.00 D.

Right eye: center thickness, 23 points; left eye: center thickness, 24 points (5 points equal 1 mm.).

These four factors contributed to the larger image of the left eye. This case illustrates a successful surgical result but a successful refractive result only after the factors causing aniseikonia had been corrected. For further consideration of these four factors it is obvious that transposition of a prescription by the optician may change the effective dioptric power and the power and size magnifications. The following case illustrates this fact:

Case 3. — K. F., a girl aged 15, had had repeated refractions but still had asthenopia. She was wearing orthogon lenses with the following correction:

Right eye: 3.25 D. sph. ⊃ 3.75 D. cyl., axis 95, on a — 10.12 D. base curve.

Left eye 2.50 D. sph. ⊃ 2.75 D. cyl., axis 80, on a — 9.12 D. base curve.

After a careful manifest and cycloplegic refraction the correction was changed to:

Right eye: —4.25 D. cyl., axis 5

Left eye: —3.75 D. cyl., axis 170

The optician was directed to grind the lenses on + 1.25 D. outside curves, with the cylinders inside. The patient was comfortable with this correction ground in this way.

There is no question of the place and of the importance of aniseikonia. Without its careful consideration refraction is not the meticulous examination it should be. Professor Ames's exposition of the subject is awaited with pleasure.

Dr. Eric Liljencrantz, Washington, D. C.: My interest in aniseikonia lies in its possible importance for aviators. It is tautological to state that space perception is of critical importance in the handling of aircraft and that any data which add to existing knowledge concerning space perception are likely to be of great importance indeed.

It is also tautological to state that views with regard to selection and training of aviators must be of highly pragmatic nature and that the luxury of academic speculation in making practical decisions regarding fitness for flight cannot be allowed. This does not mean that theory is to be ignored in aviation, but it does mean that research must be pushed to meaningful and practical conclusions. Fortunately, there has been an opportunity for just this to be done with respect to aniseikonia and naval aviation.

Thanks to Dr. Ames and his group, it was possible to examine, at the Naval Air Station, Pensacola, Fla., a large group of naval aviation cadets and experienced graduate naval aviators for aniseikonia and to compare the laboratory findings with actual performance in the handling of aircraft. The experiment was planned to answer the following questions:

1. What is the incidence of aniseikonia among naval aviation cadets selected in accordance with present naval standards? (Note: Naval standards require 20/20 vision in each eye without correction and allow little tolerance for correction with a cycloplegic or for errors in phoria.)

2. What is the effect of aniseikonia on performance in aviation?

3. What remedial steps appear to be indicated with respect to selection, corrective measures, training and the like?

I believe that the experimental data secured at Pensacola make it possible to give reliable answers to all these questions.

Dr. Robert H. Peckham, who directed the study at Pensacola, is to discuss the data. I shall anticipate his remarks by raising the following questions for further research:

1. Does such aniseikonia as is found among naval aviation cadets fail to impair their flight performance (a) because of its trivial degree, (b) because of compensatory mechanisms or (c) because binocular cues are of relatively little importance in the handling of aircraft?

2. Is the absence of subjective complaints attributable to aniseikonia among naval aviation cadets due (a) to the trivial degrees in which it occurs or (b) to the fact that aviators as a group show significantly fewer neurotic traits than do less highly selected groups?

Dr. Robert H. Peckham, Pensacola, Fla.: In order to discuss the experiments performed at Pensacola, I must point out that what I say will express my own ideas and not necessarily those of the Navy.

At the time I was doing the work, I was employed at Dartmouth College, as a civilian. The experiments were all concerned with what Dr. Lancaster has so accurately called normal aniseikonia, and my fellow workers and I were little concerned with anomalous aniseikonia and were not concerned with the pathologic conditions requiring the use of aniseikonic correction.

We examined 252 aviation cadets and instructors with the instruments available and found, first, that the eikonometer, which two years ago as the standard instrument for measuring aniseikonia, revealed no aniseikonia of a great amount. Ninety-two per cent of the subjects showed less than 0.5 per cent aniseikonia on the eikonometer, and 8 per cent showed more than that. Three per cent showed more than 0.75 per cent, and 1 subject showed more than 1 per cent —1.25 per cent.

Thus, the first thing we noticed was that aniseikonia as shown by the eikonometer was not present in the carefully selected men at Pensacola.

In addition to the work with the eikonometer, we made a series of studies on the space perception of cadets. The first study concerned the response of cadets to a frontal plane set before them and aniseikonic lenses placed so that the plane would be distorted. Their reports on the distortion were the criteria.

The second study concerned the so-called leaf room, which is a room approximately 10 feet (305 cm.) square. The subject stood so that he was in the center of the front area of this room. Questions were asked of him concerning the squareness of the room, which was accurately square, and aniseikonic lenses were placed before his eyes so that the room would be distorted.

His responses to the distortion were the criteria of his passing or failing in this test. As Dr. Liljencrantz has said, there are certain criteria of performance in this experiment. Men pass, or fail to learn to fly. Those men who fail to learn to fly fail for many reasons, among which may be aniseikonia or may be failure to respond correctly to visual space orientation. That will be only one of the reasons.

Consequently, the use of aniseikonic measurements will be masked in the consideration of passing and failing men on the basis of aniseikonia.

Thirty-three failures were carefully examined on the apparatus available, and 57 men known to be of excellent flight ability were examined, among the total. These two groups showed no differences in the relative amounts of aniseikonia as found on the eikonometer. Therefore, as far as the eikonometer was concerned, no differences could be shown between men who learned to fly well and men who could not learn to fly.

On the space perception apparatus used, there was a significant difference between these two groups. The nature of that difference was such that statistically we could say that there were 98 chances in 100 that there was a difference between the two groups on the basis of their space perception. Therefore, it is a good bet to say that binocular space perception is intimately concerned, in some cases, with flying.

Now to take the questions that Dr. Liljencrantz has raised. First, as to the incidence of aniseikonia among naval aviation cadets: Aniseikonia as found by the eikonometer was not present in a significant amount. It remains to be proved that space disorientation similar to that discovered in aniseikonic disorientation is a reliable means of discriminating between men, but there is no doubt that men showing space disorientation do have trouble in flying.

The third question suggested that remedial steps be taken. This question must be considered in the light of the two natures of the aniseikonia measured. No immediate use is to be found in measurements with the eikonometer. There is, undoubtedly, considerable work to be done with the problems of space perception, and it is possible that a careful measurement of performance and a separation of faults with respect to binocular space disorientation will be useful in the selection of aviation candidates or, at least, in their training.

If, for example, a student shows errors in phoria whose nature was predicted by his space disorientation, his instruction could be changed quickly to prevent the development of dangerous aviation habits.

As to the questions concerning further research that Dr. Liljencrantz has brought up: First, does the aniseikonia found fail to impair flight performance. If anisei-

konia as measured by the eikonometer is the criterion, there is no proof that it demonstrates poor performance. If the aniseikonia found is that associated with poor space perception, or, conversely, if poor space perception is a measure of aniseikonia, it does impair the flight performance to a statistically reliable degree.

The question of the importance of binocular cues in flying is not yet answered. One always hears of Wiley Post, who flew around the world, and then, with the same eyes, bashed into a mountain. I can see no statistically reliable evidence that binocular cues are either necessary or not necessary. They are probably of assistance if they are present and useful.

The absence of subjective complaints attributable to aniseikonia again raises the problem of the nature of the aniseikonia being measured. There was no aniseikonia as shown on the eikonometer. There was a spatial disorientation which might be aniseikonic. This was found in 33 per cent of the men examined. Thirty-three per cent of the 250 men showed spatial disorientation of moderate amounts, but amounts that might be aniseikonic.

One must say, therefore, that the failure to find subjective complaints may have been due to the significantly fewer neurotic traits in the highly selected cadets. It may be that aniseikonia as measured by the eikonometer is an index of the neurotic condition of the subject.

It requires more than mathematical analysis and the reasonableness of argument to show that binocular disorientation is dependent on aniseikonia. Nevertheless, there are sufficient evidence and proof in Dr. Lancaster's paper to indicate that experiments must be carried out in a statistical manner to show the relation between space disorientation and aniseikonia.

Dr. Walter B. Lancaster, Hanover, N. H.: I want to thank Lieutenant Peckham for his discussion of this subject. I think he must have made it clear, first, that failure to be a good flier depends on a multiplicity of causes, some ocular and many not ocular. Therefore, a statistical analysis of one cause cannot be expected to show a 100 per cent effect. Nevertheless, it is significant that the statistics he compiled after completing the experiments showed 98 chances in 100 that the distorted perception of space is a factor in poor flying.

I think it must be kept clearly in mind that in a comparatively new subject a great many uncertainties and problems are bound to arise. When a piece of research reaches the stage where there are no problems, it has reached the end of its usefulness.

The significant thing to me in the study of aniseikonia is that one after another of the problems that were puzzling are being solved, that the criticisms justly raised are, in many cases, though not in all, being answered.

This, I think, is a fair record for any new and important research.

A Simple Quantitative Test for Acuity and Reliability of Binocular Stereopsis (Lantern Demonstration). Fred H. Verhoeff, Boston

A test for grading the acuity of binocular stereopsis in which misleading size differences make binocular parallax the only correct evidence of relative depth and cause perception of false depth when this correct evidence is not perceived. This test satisfactorily determines that the subject can rely on the maximum acuity found. The grading system adopted is analogous to the Snellen system for grading acuity of vision. The usual subject has less than one chance in five million of passing the test entirely by guessing, and even a practiced cheater less than one chance in sixty-five thousand. The test is made without the use of any viewing instrument. The test seems to be the best now available for determining, in regard to stereopsis, a subject's fitness to undertake aviation or any other vocation in which high acuity and reliability of stereopsis are of great importance.

Discussion

Dr. M. C. Wheeler, New York: There are few subjects that are of greater interest and importance to ophthalmologists today than the one that Dr. Verhoeff has presented. While tests for binocular stereopsis have wide applications, their use in the selection of candidates for the flying services eclipses all others at this time.

The Howard-Dolman apparatus is too bulky for the office of the average ophthalmologist, who has been forced to rely largely on stereoscopic slides for testing binocular stereopsis and its acuity. Dr. Verhoeff's device should fill an important need, for, in addition to being superior to all other devices, it has the distinct ad-

vantage of being compact and simple to operate.

I should like to raise one question. Dr. Verhoeff has pointed out numerous theoretic objections to the Howard-Dolman apparatus, which his device corrects, yet I do not believe that he has given any evidence that the results obtained by his method, at distances under 2 meters, will be valid at distances of 6 meters or more. These are the distances at which a pilot must make his accurate estimations of distance. I fully expect that after the proper correlation with actual experience has been made, Dr. Verhoeff's method will prove to be everything that he claims, but from the evidence presented I wonder if his final statement is justified, namely, that "It seems to be the best now available for determining in regard to stereopsis a subject's fitness to undertake aviation . . ." In spite of its many deficiencies, the Howard-Dolman apparatus has proved its value on the flying field.

Dr. J. B. Hitz, Milwaukee: There has long been a need for a simple, quick, practical test of depth perception which does not utilize the principle of the stereoscope. This instrument, in Dr. Verhoeff's own words, "often seriously interferes with stereoscopic perception."

Dr. Verhoeff's instrument is as simple in construction as it is in operation. Any one with a pair of scissors and a knife can make a good working model in two hours.

My associates and I made a model instrument, following the author's directions, and tested 40 patients with it, at the same time recording the visual acuity, the ocular muscle balance at 6 meters and at 0.3 meter according to the screen test, the near point of convergence and the depth perception according to the Howard-Dolman apparatus.

We encountered some difficulty at first in getting patients to understand what was wanted from the test. This, I believe, is a criticism more of the examiner's command of English or the patient's ability to understand it than of the test itself. Occasional patients would appear at first to judge the depth difference on the basis purely of size of the object, a moment or two later perceiving the true depth difference. In all probability such patients viewed the instrument at first with monocular vision, binocular vision coming into play a few seconds later.

Since it has been well proved that accommodation plays little or no part in depth perception, at least for depth differences as small as 2.5 mm., the use of the test at from 0.5 to 2 meters is in no way an objection.

We were interested in comparing the results of actual tests with the Howard-Dolman apparatus, as this is the only instrument in general use which attempts to grade depth perception quantitatively without the use of the stereoscope.

In using the Howard apparatus we have for some years used Howard's original method, that of having the examiner set the targets under cover, the patient only stating which rod is nearest to him. In order to record a patient's depth perception at a given separation of the rods, fifteen consecutive correct answers are required. This number of checks gives the examinee less and one chance in 34,000 of getting all the answers correct by guess work. The choice of whether the movable rod is in front of or behind the stationary one is left to mathematical chance, shuffled cards being used to determine its position. We used this method rather than that of allowing the patient to adjust the apparatus himself because, first, it is far simpler to understand, particularly for children, and, secondly, we desired to find the minimal separation at which the patient could constantly judge depth rather than the maximum at which there was no depth discernible.

In comparing the two tests, we found that a comparison of the actual parallactic angles was not possible because of the marked increase in angle for a given depth difference when the distance from the instrument to the eyes was lessened. Therefore, we selected arbitrarily 20/25, or the ability to detect a 2.5 mm. depth difference at 80 cm. on Dr. Verhoeff's instrument and a 35 mm. separation at 6 meters on the Howard apparatus, as criteria for passage of the tests. Interpreting our findings on this basis, we found that of our series of 40 patients (1) 30, or 75 per cent, passed both tests, (2) 6, or 15 per cent, failed both tests and, (3) 4, or 10 per cent, were inconsistent. Of the 4 last-mentioned patients, the condition of 2 was borderline and 1 had a definite mild myopia accounting for failure in the 6 meter test and success in the 1 meter test. In other words, for the sake of discussion the criteria mentioned being accepted for passage or failure of the two tests, there

was a definite correlation in the findings in 90 per cent of the cases.

Calculating the parallactic angles for the two tests (omitting 4 cases in which depth perception was so poor it could not be recorded), we found that the parallactic angles subtended in Dr. Verhoeff's test averaged slightly over four times those in the Howard test. I should like to ask Dr. Verhoeff whether he accounts for this difference by principle of size difference introduced in his test.

Other interesting points in this study might bear further investigation: While there were 8 patients who showed defective vision, heterophoria or poor convergence and passed the depth perception tests, no patient who had normal vision, muscle balance and convergence according to the generally accepted criteria failed to pass the depth perception tests. This would suggest the fact, which I have believed to be true for some time but which I have not seen stated in the literature, that with normal vision and normal extraocular muscle function as tested by accepted methods, poor depth perception is a rare phenomenon. Other factors have been mentioned in the literature which could have an effect on depth perception, namely, (1) hyperopia of sufficient amount to disturb the accommodation-convergence relationship and (2) aniseikonia.

Dr. Verhoeff has presented an extremely valuable instrument which is simple and inexpensive to construct, is quick and easy to use and compares favorably with other instruments, which, while they may not be perfect have stood the test of time as practical and hitherto indispensable instruments.

Dr. Karl W. Ascher, Cincinnati: Binocular instantaneous depth perception does not range gradually from a normal average to zero, as visual acuity does. When a large number of persons are tested by the same method, the thresholds of stereopsis will be close to each other provided visual acuity and muscular balance are normal. While the influence of auxiliary motives may create boundless differences in the judgment of distance, depth perception due to binocular dissimilarity of retinal images is either present or absent. Its presence can be easily proved by the drop test of Hering. Peas slightly varying in size and falling at a minute distance beyond or in front of the tip of an immovable needle are exposed for fractions of a second. With this test, a 100 per cent of correct answers will be obtained from persons with normal stereopsis. With Dr. Verhoeff's ingenious apparatus, too, the thresholds of numerous persons previously selected for good visual acuity and muscular balance will probably have almost equal top values owing to the fact that the threshold for stereopsis corresponds closely with the threshold for vernier acuity.

Up to this time the methods for testing of depth perception have been based on the highest possible exclusion of auxiliary motives of space localization. In practice, however, just these secondary motives of depth judgment are the only ones available at the distances concerned in practical flying. The accuracy of retinal binocular depth perception drops considerably in the peripheral field, and it falls at an increasing ratio with the absolute distance of the observed object. While, at a distance of 1 meter with a threshold of 5 arc seconds, 0.5 mm. differences are exactly recognizable, at 1,000 meters, only a difference of 274 meters can be appreciated. For these distances, only auxiliary motives of space judgment are of value. Accommodation and convergence, not very efficient at close distances, are of no help whatever at the distances important for flying. The auxiliary motives of distance judgment may vary considerably, while the differences in the threshold of binocular stereopsis are more dependent on the method used and on the differences in function from person to person provided there is normal vision. I wonder whether a flier attaining 65 per cent in the new Keystone test would be more exact in landing than one with only 60 or 55 per cent.

The evaluation of the auxiliary motives in distance judgment can be obtained by a method based on the fact that equally sized squares exposed at different distances appear to be equal only when their relative and their absolute distance are correctly evaluated by the tested persons. With the utilization of fewer auxiliary motives or with insufficient utilization of them, the influence of the size of the retinal image will increase and the judgment about the apparent size will be led principally by the visual angle. The square comparison test is the only possible test for space judgment for distances beyond 1 mile (1.6 kilometers).

I should like to recommend the square comparison method for examinations of aviators for four reasons:

1. It includes all motives of distance judgment in a subconscious way.
2. It can be used in the central parts of the visual fields as well as in their periphery and even in the temporal crescents of monocular vision.
3. It concerns just the distances at which dissimilarity in the retinal images ceases to be of value.
4. Dissimulation is improbable.

Dr. Walter B. Lancaster, Hanover, N. H.: I wish to discuss Dr. Verhoeff's test from what the lieutenant spoke of as the pragmatic point of view: not whether this is a good test for stereopsis but whether granted the test is good, it is of value to know the depth perception, for example, of an aviator or of a man who is handling machinery.

Stereopsis gives a keen and accurate estimate of the relative distance of two objects which are fairly closely juxtaposed and not far off. By relative distance I mean the distance of one compared with that of the other. If I look at two pencils side by side rather close together, at a distance of a meter or two, stereopsis will enable me to tell whether one is a little farther away than the other (relative distance); but if, instead of being close together, they are widely separated (say 0.5 to 1 meter apart) but still a meter or two from the eyes, stereopsis cannot provide accurate judgment as to which is nearer the eye. Moreover, as to how far both of them are from the person who is looking (egocentric factor of space), stereopsis does not give that kind of important information.

It is not often that persons have trouble with stereopsis when they have good visual acuity and are not suppressing the image of one eye or otherwise avoiding binocular vision. I think the most impressive way to demonstrate this is to show that a person can have considerable abnormal spatial perception and still get perfect stereopsis. With glasses which cause a considerable and obvious distortion of space, one gets perfect stereopsis if one had it to begin with. In other words, stereopsis, as some of the armed forces have decided, is of little value in an estimation of an aviator's capacity for accurate spatial localization.

Dr. Morris Davidson, New York: I should like to call attention to the usefulness of quantitative depth perception. It is not true that a person either has depth perception or has not got it. A person who has one eye, of course, has not got it. Persons who have two eyes and normal visual acuity in either eye have varying depth perception. What the variations are due to is too difficult to analyze in brief. But one might say that just as normal eyes have visual acuity varying between 20/20 and 20/10, so normal pairs of eyes have a stereoscopic acuity varying between certain limits.

I should also like to point out that there are monocular clues to depth judgment. Therefore, there is a threshold of monocular depth judgment. The smaller the distance at which one tests, the more likely there is to be an approximation between the high threshold of binocular depth perception and the low threshold of monocular depth judgment. Therefore, at less than 2 meters all tests become unreliable.

The optimum range of testing is between 2 meters and 6 meters. I do not believe that the Howard-Dolman method is bad. I think it is an excellent method and based on sound, scientific, physiologic principles.

What is the normal? By practically all methods based on sound, scientific principles, 20 seconds of arc is normal stereoscopic acuity. That is present in 60 per cent of persons with two normal eyes. Therefore, it may be considered as normal. But the quantitative test becomes inaccurate at any distance less than 2 meters, and I do not believe that it should be used at that distance or less if one wishes accurate results.

Dr. Robert H. Peckham, Pensacola, Fla.: A point that has been brought to mind regarding the problem of steroscopic perception with relation to aviation or any other activity requiring binocular vision is the basic importance of the work at Dartmouth.

Binocular visual orientation includes more than the measurement of a small central area surrounding the point of fixation.

The performance problems required in aviation and in almost every other form of activity in which binocular vision is necessary involve the use of the entire peripheral field. Consequently, a test is

necessary for stereopsis, but that test must concern itself with the response as it pertains to the whole field.

The Howard-Dolman test is an accurate, reliable test for the small, central portion of the visual field. The test which Dr. Verhoeff has described is a more reliable, more accurate test for performance in that same small and relatively unimportant area. The major problem is to discover by what means, both eyes being used, one's orientation of oneself with respect to space is accomplished.

I bring the point up in this discussion because I have seen so many tests devised for central stereopsis. There are, perhaps, forty or fifty, and if I had nothing else to do I could devise another forty or fifty. Some of them would not be as good, and most of them would be much poorer, than the one Dr. Verhoeff has presented. But to ophthalmologists who feel it necessary to make a test, let me make a plea that they make a test to determine spatial orientation requiring the use of both eyes.

Dr. F. H. Verhoeff, Boston: My test determines the subject's ability to interpret binocular parallax as relative depth. It seems to me that this ability should be the same at any distance. However, the less the distance at which the test is made, the more importance binocular parallax has over relative size.

As I understand it, Dr. Hitz modified Howard's original test and then compared it with mine. Since the conditions still differed from those in my test it would be expected that the two tests would differ as to the minimum perceptible binocular parallax. This makes evident the importance of standardizing my test. Under certain favorable conditions a binocular parallax of less than 1 second can be recognized as depth, but it would not be practical to employ such condition in a clinical test.

Dr. Lancaster discussed the difference between absolute and relative depth. My test is for relative depth. He said that the armed forces have found that the test of stereopsis is of little value. He should have said that they have found the Howard-Dolman test to be of little value.

Dr. Ascher brought up the old test of Hering, the falling ball test. This test as ordinarily made is open to serious objections even as a qualitative test and certainly cannot be relied on as a quantitative test.

Experimental Studies on the Dynamics of the Intracapsular Cataract Extraction, Suspensory Ligament and Hannover's Canal (Lantern and Motion Picture Demonstration). Jacob Goldsmith, New York

The details of the physicodynamics of the intracapsular cataract extraction were viewed through the corneal microscope from various aspects on differently prepared specimens and a study was made of the effects on the anatomic structures in the anterior segment of the eye during the course of this procedure. The contents of Hannover's canal were aspirated and examined. The walls of this canal were stained and proof presented that the zonular bundles are transformed into a continuous membrane, anteriorly as well as posteriorly. The suspensory ligament in its entirety was investigated from its origin on the pars plana up to its lenticular insertion (zonular lamella of Berger). Serial microscopic sections were made from a normal human adult eye, and another series from a normal adult eye that had undergone an intracapsular extraction.

Discussion

Dr. Manuel Uribe Troncoso, New York: Dr. Goldsmith's present paper is a more complete presentation of the subject than was his former article in the Archives (Arch. Ophth. 22:792, Nov., 1939.) In this new work, besides the study of intracapsular extraction by suction, he considers, first, the removal of the lens with a capsular forceps aided by external pressure and, second, the Smith (Indian) operation with external pressure alone.

I am glad that he used to advantage the anatomic method with the slit lamp microscope that I recommended in 1936. This gives a much more accurate stereoscopic view of the architecture of the zonula and surrounding structures than is obtained by simple microscopic methods, in which the section is never a complete aspect of the organs. About the technic of dry preparations, I may say that it has the defect of prolonged exposure to the air, which dries and changes the tissues. I have found it preferable to make my dissections under water, because in this way the zonula, lens and processes stay in position and have normal relations. In some cases I use the ordinary dissecting microscope to advantage, with the specimen under water and in a downward position.

Dr. Goldsmith's paper sets forth clearly the dynamic factors in play both internally and externally during intracapsular cataract extraction.

He had the rare fortune of securing 59 fresh human eyes from four to six hours after death and was able to visualize the changes brought about in the zonula and the ciliary body by traction and pressure. He emphasized the complete independence of the vitreous from the posterior zonular membrane downward and also the independence of the zonula from the ciliary processes in their forward two thirds. He has studied the close adhesion between the lens and the hyaloid fossa. His experiment of cutting down the zonula with scissors and then trying to remove the lens with a capsular forceps showed him how strong is the resistance of the capsular adhesion to the patellar fossa owing first to negative pressure and second to the existence of the ligamentum hyaloideocapsulare. The force of this resistance is important to consider during intracapsular extraction.

His researches on the Smith (Indian) method, in which the zonula is torn in the lower part of the lens by heavy external pressure on the scleral surface with a hook, show that it has the effect of pressing the ciliary processes against the equator of the lens, which displaces the zonula back laterally almost 80 or 90 degrees and ruptures it. Then the lens is subluxated upward. This maneuver puts the superior ciliary processes under tension and twists the lateral zonular bundles. In order to overcome the resistance, a much greater pressure is necessary, which leads sometimes to prolapse and loss of vitreous. He does not recommend this method of extraction, which produces a great deal of trauma.

Use of the capsular forceps with lateral to and fro movements of the lens causes an inferior rupture of the zonula. When helped by external pressure with a hook applied to the sclera it is safer than the Smith method. Instead of the forceps the suction cup is also convenient. However, it is not only the rupture of the zonular fibers which is important, but the tumbling of the lens by which the freed lower portion of the equator is tilted upward and outward, rotating anteriorly 180 degrees.

Dr. Goldsmith has shown that the greatest factor in the dislocation of the lens is the side to side shaking movement, while the backward pressure acts over the posterior spaces, the lens and the zonular diaphragm, helping to produce the tumbling. The pressure of the hook acts by hydrostatic transmission of the force backward and upward, which helps to separate the lens from the fossa.

Thus research work has supported the surgeon's view that the Smith (Indian) method is rough and should not be employed, while the operation with the capsular forceps or the suction cup accompanied by external pressure increases the probabilities of success and produces much less trauma to the zonula and the ciliary processes.

Dr. Goldsmith has taken a great deal of trouble to prove the existence in man of Hannover's canal. This canal is not present in rodents, ungulae and carnivora, in which the zonular fibers form a solid bundle inserted over and to each side of the equator of the lens. In primates the anterior and posterior zonular membranes are inserted separately into the anterior and posterior lens capsule over the equator and leave between them a triangular space, whose apex is attached to the ciliary processes at the valleys. This space is easily seen in meridional sections, but it is not a continuous circular canal. It is partitioned irregularly by many isolated bundles of zonular fibers, which serve mainly to suspend the lens. If one admits that the anterior and posterior zonular layers are continuous membranes, then one must admit that Hannover's space is a closed, irregular, circular cavity, with its base over the equator of the lens and its apex where the two zonular membranes join and fuse at the level of the posterior third of the length of the processes.

Dr. Goldsmith has confirmed by chemical examination of the fluid aspirated from this space the conclusions of previous authors (Beauvieux, Duke-Elder) that there is a viscous liquid in Hannover's space. This fact is important from the physiologic standpoint.

Dr. Henry J. Minsky, New York: Berger in 1887 noted that Wieger and Virchow disagreed over Petit's canal. Today, Dr. Goldsmith and I, working independently in one institution, have come to a similar controversy. I cannot agree with many of Dr. Goldsmith's conclusions, because the conditions of his experiments on intracapsular extraction are not those of the

actual operation. Time permits me to discuss only four differences of opinion:

1. The zonular membrane does arise from the same source as the vitreous. In this 112 mm. embryo (slide) Dr. Kornzweig found the zonular fibrillae (tertiary vitreous of Ida Mann) morphologically indistinguishable from the vitreous.

2. The hyaloid membrane does contain zonular fibers. Retzius in 1894 showed the zonular fibrillae splitting into two layers about 2 mm. from the ora to form Petit's space (slide). One layer ran to the ciliary epithelium, while the other (called the central layer by Graf Spee and the innermost fibers by Salzmann) arched over the hyaloid membrane. This real space between the two layers (which should be called the zonular chamber) is different from that of Petit, who described a potential space between hyaloid membrane and zonula extending from lens to retina. The insertion of zonular fibers into the hyaloid membrane is demonstrated when the isolated lens and vitreous body are suspended by sutures taken in the suspensory ligament (motion picture). Negative pressure plays a minor part in the adherence of the lens to the hyaloid membrane. Finally, I have demonstrated the hyaloid-zonular fibers as brilliant white lines in a purple field by double-polarized light in unstained sections mounted in 70 per cent alcohol. Balsam masks the visibility of the zonular fibrillae and accounts for their obscurity.

3. The architecture of the suspensory ligament is like a double shell dome braced by cross members. In the periphery, the hyalozonular leaf is more prominent than the ciliary fibers (slide). It tapers centripetally by losing bundles to the ciliary portion. Those hyalozonular fibers which bind the ciliary processes to the hyaloid membrane run through Hannover's canal to the anterior capsule. The ligamentum hyaloideocapsulare can be seen microscopically, since it is the termination of the zonular lamellae of the hyaloid membrane on the posterior capsule.

In intracapsular operations, pressure should be applied near the equator of the lens, where the shearing action of the communicating bands is at a maximum (slide). Although the force is absorbed mainly by the ciliary processes, excessive pressure pulls the retina and the vitreous. From an experimental approach, the intracapsular operation involves more anatomic structures and invites more physiologic disturbances than the extracapsular.

4. Hannover's canal is not a closed space, but a subdivision of the posterior chamber for descriptive purposes. When Dr. Goldsmith removes the cornea and iris and substitutes air for aqueous around Hannover's canal, he loses the normal interfaces and increases the surface tension, so that the space fills by capillarity in spite of fenestrations in its "walls." A molecular dye injected into Hannover's canal under conditions approaching normal fills the anterior, posterior and zonular chambers (motion picture).

Dr. F. H. Verhoeff, Boston: The most important thing Dr. Goldsmith did was to aspirate the Hannoverian canal and find it contained a fluid that was albuminous. If the canal will retain albuminous fluid, of course it does not make any difference whether one finds other evidence that there is membrane around it. As to the observations that were made by Dr. Minsky, it seems to me that if a fluid is injected forcibly anywhere it may break through a membrane. His observations therefore do not afford conclusive proof that there is not a thin membrane around the Hannoverian canal. In his experiments Dr. Goldsmith used cadavers. In postmortem specimens protein material soon dissolves in the surrounding fluid, and one gets albuminous fluid. I think he may have controlled his experiments—he did not say. He may have done so by aspirating the fluid from around the Hannoverian canal, the posterior chamber and the aqueous to see if that fluid was any different from the fluid in the canal. If it was different, I think he has made an important contribution.

Dr. Goldsmith spoke about making a microscopic examination of a normal eye. I did not hear him mention it, but I know he is aware of the fact that about ten years ago I performed an intracapsular extraction on a freshly enucleated normal eye and then examined the eye microscopically. I found that no damage had been done to the retina or the ciliary epithelium. The zonular fibers had ruptured near the lens. As far as I know, that was the first time such an experiment had ever been done. I performed the same experiment on each of 2 eyes which were not entirely normal. One of the eyes had fluid vitreous. The operation produced no damage to the ciliary epithelium or to the retina in either eye.

Dr. J. Goldsmith, New York: In reply to Dr. Troncoso I wish to state that in my experiments the zonular membranes were continuously bathed with solution, to avoid drying of the fibers. Tumbling, other internal procedures and the application of external force introduce factors of stress and strain, with the liberation of energy, which is constantly being dissipated into the surrounding fluid medium. This prevents damage to the neighboring delicate structures. I believe the study of Hannover's canal is important. Since Liesegang rings were clearly demonstrated in the aspirated contents, a colloidal system must exist in this canal. A modification in the chemistry of this complex system most probably alters the nutrition to the lens, the alteration eventually initiating cataractous changes. Dr. Troncoso stated that he found degenerative changes about the lens equator (involving the zonular membranes) in studying eyes which had incipient lenticular changes. Time does not permit me to discuss the controversial points raised by Dr. Minsky. Each point is taken up separately in my paper. In 1922 Beauvieux claimed that the suspensory ligament is separate and independent of the hyaloid membrane, anatomically as well as embryologically. The marginal bundle of Druault (secondary vitreous) atrophies, and its outermost surface forms the future hyaloid membrane. The anterior and posterior zonular fibers which are laid down anteriorly to the hyaloid membrane are separate and independent of it.

The zonular chamber described by Dr. Minsky corresponds to the well known space of Petit's canal. Petit described the superoposterior extremity as commencing at the ora serrata. Ivanoff placed it at 4 mm. and Wieger at 2 mm. from the ora serrata. Hannover's work confirmed Petit's findings. My recent studies place the closed posterior extremity at 3 mm. away. The only space to be rightfully called the zonular chamber is Hannover's canal.

Anatomically there is no proof to substantiate that the vitreous is suspended from the lens by the zonular membrane. The anterior and posterior zonular membranes are extremely delicate, and any manipulation, no matter how slight, especially insertion of sutures, would be sufficient to tear it immediately.

In regard to Dr. Verhoeff's question, I should like to state that examination of the aqueous (prezonular space) revealed no Liesegang rings. Study of the vitreous (postzonular space) showed it to abound in Liesegang rings; this finding was ably demonstrated by Redslob in 1932. The vitreous body was completely separated before Hannover's canal was aspirated.

The results of study of serial microscopic sections of eyes which had undergone intracapsular extraction confirm those obtained by Dr. Verhoeff in 1931. There was no damage sustained, either grossly or microscopically, to the hyaloid membrane and the epithelium lining the ciliary body.

It has been possible by radiographic technic to demonstrate Hannover's canal and Petit's space as separate and independent chambers. Air, barium sulfate, 20 per cent sodium iodide, and fine metallic bodies were injected into the spaces. Lateral, oblique and anteroposterior views were taken on dental films.

I thank Dr. Troncoso, Dr. Verhoeff and Dr. Minsky for their participation in the discussion.

Color Discrimination in Industry (Lantern Demonstration). Joseph Tiffin, Lafayette, Ind., and Hedwig S. Kuhn, Hammond, Ind.

Summary of experimental data based on survey results of approximately seven thousand industrial employees, which show that (a) color vision apparently is important for certain industrial jobs that have not been identified from logical job analyses as requiring color vision, (b) color vision is markedly reduced with increasing age and (c) color vision exists in greater or less amount on a continuous scale from color blindness at one extreme to very keen color sensitivity at the other. The evidence leading to these three conclusions, and the industrial implications of this evidence.

Discussion

Dr. Alfred Cowan, Philadelphia: This investigation by Dr. Tiffin and Dr. Kuhn has certainly brought out some surprising results. My first reaction is an intense desire to learn the method of examination, based primarily on red-green discrimination, by which such fine determinations can be made. It is well known that what is generally spoken of as partial color blindness exists in varying degrees, probably similar to the varying degrees of visual acuity, and probably if there were some means of measurement color vision could be noted as visual acuity is noted. But as yet there is no clinical meth-

od of testing color vision which is comparable to the tests for visual acuity.

By the unrefined office methods ordinarily used for testing color vision on the basis of red-green-blue discrimination, the best that can be stated is that a person is a monochromat, a dichromat, an anomalous trichromat or a normal trichromat. It will be extremely interesting to learn of a reliable test of the color vision, based on the Young-Helmholtz theory, consisting of four items of which the most difficult would screen out 44.7 per cent. I am sure most ophthalmologists will agree with me that according to the current conception this is an extremely high percentage of color defectives. It seems, too, that if the same results would hold generally, relatively few persons would qualify as examiners. Also, while it is known that color discrimination is reduced because of certain senile changes which take place with increasing age, it seems remarkable that the authors have found that at the age of 45 the percentage of failures has already risen to about 57 per cent, as shown in chart 2. These figures are somewhat upsetting in view of the fact that almost without exception master dyers are well past middle age.

Of greatest importance, however, is the conclusion of the essayists, after analyzing their data, that they have devised a practical means of estimating the aptitude, efficiency and intelligence of workers in every type of industrial job by the application of their four item test of the color sense.

Dr. Peter C. Kronfeld, Chicago: This work by Dr. Tiffin and Dr. Kuhn is primarily intended for and is of special interest to those ophthalmologists who act as advisers in matters of the visual requirements for employment in industry.

The study has brought to light at least two findings of general ophthalmologic interest, namely (1) a higher incidence of anomalies of color sense than has been reported in other populations and (2) a dependence of this incidence on the age of the employees.

In order to be able to evaluate these findings, one has to know a little more about the test which in the paper, is described as "a simple four item red-green discrimination test."

The test was conducted with cards put out by the Keystone Company and apparently designed on the same principle as the pseudoisochromatic plates of Stilling or Ishihara. To me the four items seem very easy, and I do not understand why almost one half of the entire group, or one fourth of the persons under 30 years of age, failed to pass the test. Peculiar lighting may have made the test more difficult. It would seem significant that if the most difficult item was omitted from the test the incidence of anomalies of the color sense would be reduced to roughly 10 per cent, a figure which is in close agreement with the results obtained with other methods in other populations.

The apparent incidence of deficiencies in the color sense depends to a large extent on how fine a test is used. Theoretically, at least, it would be possible to design a test which would be passed by a still smaller percentage of persons than that reported here.

The fundamental question, therefore, is, what degree of color discrimination is necessary for efficient performance of a certain job? If the answer to that question were available, it would be fairly easy to devise a test of adequate fineness and accuracy. For quantitative studies of the color sense of the accuracy required in the color printing industry, it would seem to me that the most accurate and dependable test should be used, and in my opinion this should be a test based on equations between mixtures of spectral red and green on the one side and spectral yellow on the other side.

The authors have stressed the quantitative nature of the ability as well as the inability to discriminate colors on a continuous scale. In principle this is true, since with accurate methods the occurrence of many gradations or transitional forms between the normal trichromatic, the anomalous trichromatic, the typical dichromatic and the monochromatic color systems have been demonstrated.

With regard to the apparent diminution in the ability to discriminate colors which occurs with increasing age, I wonder if the authors have any explanation to offer for this phenomenon. Since it occured in eyes free of any gross disease, I would be inclined to correlate it with the absorption of light of short wavelength by the crystalline lens, which occurs in the eyes of older persons to a greater degree than in young persons.

Whether or not a prospective employee in a color-printing plant is likely to acquire a marked degree of this absorptive form of color deficiency in later years

would seem to me difficult to determine at the age of 20, but that is the time when it should be decided whether the applicant is a good or a bad risk from the viewpoint of color discrimination. The degree of color sense which the applicant possesses at the age of 20 would, to me at least, not be indicative of or related to the degree of color deficiency that might result from changes in light absorption by the lens later on. Besides, such changes in the lens occur so slowly that an experienced employee could be expected to make the necessary adjustments, consciously or unconsciously.

Dr. Joseph Tiffin, Lafayette, Ind.: I should be the first to agree, and I am sure Dr. Kuhn would also agree, that we are not satisfied with this particular testing device for segregation of industrial employees, whereas we would both agree that a test which permits only 55 per cent of a large group of persons selected at random to pass it is, in general, a fairly stiff test or a fairly fine test.

We feel rather strongly that, fine as the test is, it is still not fine enough, because there are a good many types of industrial jobs (jobs calling for a high degree of color discrimination) in which one wants to know not that a person is in the best 55 per cent of the population but whether or not he is in the best 10 per cent or the best 5 per cent. It would be exactly the same as if one had a thermometer that only measured the temperature reached on 45 per cent of the days. It would be a fine thermometer for 45 per cent of the days, but on the other 55 per cent it would leave quite a bit to be guessed.

Industrial classification of employees clearly indicates that there are many cases in which one wants to measure accurately the exact degrees of color vision that the upper 55 per cent have. That is exactly what we have striven toward in the construction of a new test — which I might say by way of anticipation does definitely accomplish the desired result.

The Removal of Metallic Foreign Bodies From the Eyeball and the Orbit (Lantern Demonstration) Edmund B. Spaeth, Philadelphia

In considering this subject from a diagnostic standpoint, three factors are in need of discussion: the more extensive use of stereoscopic x-ray films, especially in extraocular foreign bodies; accurate chart localization to make more certain the differentiation of intraocular from extraocular positions; the more extensive use of corneal and scleral radiopaque fixed landmarks. In considering surgery, four procedures are to be mentioned: the use of fixed radiopaque landmarks; the biplane fluoroscope as it applies to extraocular foreign bodies and to cases of intraocular nonmagnetic foreign bodies with an opaque vitreous as seen with extensive intraocular vitreous hemorrhages; the use of the hand magnet; the ocular endoscope, which is especially valuable in those cases of intraocular nonmagnetic foreign bodies which have a clear vitreous. This consideration of the subject is rather timely.

Discussion

Dr. Edward Stieren, Pittsburgh: I do not subscribe to the statement that foreign body in the vitreous means potentially a lost eye. The vitreous body is more or less of a mystery. Duke-Elder considers it a gel. My experience is based on forty-five years of practicing ophthalmology in an industrial community with a record of more than eight hundred extractions of foreign bodies from within the eye. Long ago I noticed that if the vitreous was much disturbed or a fourth of its bulk was lost it resulted in a sick eye, an eye that was not inflamed or infected but marked by lack of the beautiful transparency of the virteous, so that the fundus would appear as though looked at through a fog. These eyes usually develop minus tension and go into decline, a great many ending in phthisis bulbi. In removing foreign bodies from the vitreous I feel that it is a great mistake to plunge any instrument, such as a cataract knife, in making the scleral section or introducing the various tips that come with the hand magnet into the vitreous and stirring it up. Probably the accident happened about an hour or two before and the wound of entrance is still open. The temptation is great to apply the magnet immediately. I advise against this for two reasons: The foreign body may not still be in the eye; it may have gone through into the orbit. We must know the size of the foreign body, as well as its location, before attempting removal. The same argument obtains against using the electromagnet as a diagnostic instrument, as a sideroscope. The foreign body may not be within the eye at all. If in the case of a recent injury it cannot be seen with the ophthalmoscope, the eye should be roentgenographed and the foreign body plotted. Then one knows

just where the foreign body is, and its size. The sclerotomy is performed with gentle dissecting strokes until the choroid presents, and then a very delicate opening is made through the choroid. Then the tip of the magnet, either the oval or the cone shaped tip, is introduced into the lips of the wound, and only then should the current be turned on. The wound of entrance is misleading sometimes. A foreign body may be long, 4 or 5 mm., and very narrow, and maybe it has left a very small opening in the globe. But in being withdrawn with the magnet it may become lodged crosswise. Instead of tugging and pulling and trying forcibly to remove a large foreign body through an inadequate opening, it is much better surgery and less hard on the eye to have the opening accommodate the size of the foreign body. Dr. Spaeth suggests a hinged flap. I have never tried that. I prefer a T shaped incision.

Dr. Raymond L. Pfeiffer, New York: Dr. Spaeth has reviewed the mechanical and surgical aids which must be available to one seeing many cases of trauma of the eye. A great deal can be said on each of these aids, and one's experience with each does not always correspond with the degree of importance which Dr. Spaeth has indicated. He rightly emphasizes the importance of meticulous x-ray work in the diagnosis and localization of intraocular foreign bodies. Many methods are available for this work, and the roentgenologist is most proficient with the method with which he is best acquainted. I have described a simple, inexpensive and accurate method which gives a meridional plotting easiest for the surgeon to utilize in the operating room. This method employs a contact lens similar to the Zeiss lens which was adapted to the localization of foreign bodies in the eye by Comberg in 1928. After the lens is placed on the injured eye, posteroanterior and lateral films are made of the orbits, and lines are scratched directly on the films to indicate the meridian in which the body lies, its distance out from the anteroposterior axis of the eyeball in this meridian and its distance back from the limbus of the cornea. The measurements, minus correction factors due to the magnified distortion which occurs in all x-ray films, are transferred to a specially prepared drawing, which shows the eye in front and lateral views enlarged three times for accuracy of plotting. This method is simple, rapid in operation and accurate. It is impossible to make a significant error with it. One need not fear placing the lens on severely injured eyes if a few drops of a local anesthetic such as pontocaine are used. This method is especially well adapted to field work. Nine years of experience in a busy eye hospital with this method has been most convincing. Soft tissue films or bone free films, first advocated by Vogt, should be made in every case of suspected intraocular foreign body. These films are easily prepared and are capable of revealing the most minute foreign bodies of any density greater than that of the tissues. Glass fragments may be revealed with them. They may reveal bodies which can be overlooked in ordinary films. Their usefulness, however, is limited to the anterior segment. In ordinary cases of metallic foreign body they offer a check on the localization achieved by other methods and reveal the shape and suggest the character of the foreign body.

Dr. Harvey E. Thorpe, Pittsburgh: It is important to make a careful slit lamp examination to determine whether there has been a penetration of the cornea, sclera, the iris or the lens. An x-ray examination of the eye is essential. The method of x-ray localization with air injection in Tenon's capsule described by Dr. Spaeth should be valuable. I use Sweet's method of localization and with it frequently combine Comberg's method. I now use a plastic contact lens instead of the glass Comberg lens. Instead of the lead markers, which are difficult to insert into the plastic lenses, I use small platinum wire markers, which can be placed accurately into the plastic contact lens. The contact lens method of localization of intraocular foreign bodies can be combined with a stereoscopic technic. This enables the ophthalmic surgeon to view the eyeball either by the air injection method or, possibly easier, by the method of the contact lens in stereofilm. I cannot stress too strongly the use of the stereofilm and accurate localization, because only then does one know where to make the incision for the removal of the foreign body. The use of the ocular endoscope is a protean procedure. I do not recommend it in the hands of some one who has not previously acquired practice on many animal eyes. It is essential to practice first with the operating end of the endoscope inserted into a little hollow sphere (1 inch in diameter). This should be followed by

practice on animal eyes to acquaint oneself with an instrument that is guided only by monocular vision. Being prepared for this major procedure, one may now attempt the removal of nonmagnetic foreign bodies from a nonbloody vitreous. I devised this instrument primarily for the removal of copper, lead or other nonmagnetic foreign bodies from the vitreous. It has been the general experience of ophthalmologists that a piece of copper in an eye meant enucleation of the globe. The use of the endoscope may save the globe and may even preserve sight. One is therefore justified in using the instrument. The endoscope measures 6 mm. across. It is not introduced beyond the scleral incision; it is placed just within and between the lips of the scleral incision. The lips of the wound are held open by previously inserted scleral sutures. The assistant holds the operating end of the instrument to prevent its being shoved too deeply into the globe while the surgeon has his eye at the observing end of the endoscope. With the miniature electric bulb illuminated, only the minute tubular forceps (1.5 mm. in diameter) is pushed down to the lead shot.

Dr. Oscar Wilkinson, Washington, D. C.: I wish to emphasize the importance of attempting to remove very small foreign bodies from the vitreous as soon as possible after the injury. When the foreign body is visible with the ophthalmoscope and one can use a hand magnet and determine, with the ophthalmoscope, whether it is magnetic or not, it facilitates matters very much more to do this early, before any inflammatory changes have taken place. Recently I deferred until the next morning the removal of a foreign body in the vitreous which was subject to magnetic influence, at which time there was no hemorrhage at all in the vitreous. The next morning, when I undertook its removal, the vitreous was full of hemorrhage and I could not find the foreign body without x-ray localization. The use of the hand magnet in removing smaller objects is superior to the larger magnet. With the larger magnet one sometimes gets the foreign body and the eye too. With reference to the scleral route, several years ago I heard Samuels discussing the effects of foreign bodies on the vitreous. His idea at that time was that a foreign body in the vitreous meant that the vitreous would be so altered and so mutilated that eventually the sight would be practically lost. If these minute foreign bodies are removed immediately, the prognosis is more often favorable. One saves a man's eye and gets practically no reaction afterward. I always use fulguration to prevent hemorrhages in making the scleral incision. When a patient comes into one's office with the history of an injury, practically always a penetrating wound will leave a lowered tension of the eyeball. It is a good plan to put one's finger on that eye, and if it is softer it is an indication that that eye has been perforated.

Dr. Elbert S. Sherman, Newark, N. J.: For the successful management of a case of intraocular foreign body proper equipment, experience and good judgment are important. For years there has been discussion as to whether it is safer to remove a piece of steel from the vitreous by the anterior route or through an incision in the sclera. In many cases, for well known reasons, the anterior route should be chosen. In others, probably a small majority, the transscleral route is safer and easier. It has been alleged that detachment of the retina is a frequent sequel of removal by the posterior route. As I have shown (Am. J. Ophth. **22**:1368 Dec., 1939) retinal detachment is not a necessary complication. In my experience it has occurred only once. This may be because most of the cases have been seen promptly and operation performed within twenty-four hours after the accident. Another important reason is that no instruments are permitted to enter the vitreous, and this vulnerable structure is disturbed as little as possible. If done early, before the eye has become inflamed or adhesions have formed, the operation is easier and complications are less likely to occur. I have had no experience with the biplane fluoroscope but have been unfavorably impressed by the late results following its use. I agree with everything that Dr. Stieren has said.

Dr. Stieren: In a paper read before the American Ophthalmological Society in 1935 Jonas Friedenwald presented a diagrammatic drawing showing the arrangement of the sheets comprising the vitreous. It illustrates what happens when one introduces anything into the virteous chamber. Friedenwald does not say so, but it seems to me that this viscid ma-

terial, the fluid between these sheets, if it is not lymph, must act as lymph and is intended for the nourishment of the inside of the eye. When one destroys these sheets one destroys the channels.

Dr. Edmund B. Spaeth, Philadelphia: Dr. Pfeiffer has brought to our attention the importance of the x-rays in the handling of these cases. Relative to Dr. Sherman's statement, there is now a patient in the hospital who emphasizes this matter of retinal detachment. A foreign body perforated the sclera 10 or 12 mm. behind the limbus. The patient on admittance to the hospital had a trifoliate retinal tear and at the end of three days a complete retinal detachment. The patient was treated from the time of his admission to the hospital with absolute quiet and pinhole glass, but in spite of this, detachment occurred. With regard to the use of the endoscope, it is absolutely necessary that these cases be taken care of very early; otherwise changes which occur in and of the vitreous make it impossible even to grasp the foreign body with the endoscope forceps, because of the infiltration and condensation of the vitreous encapsulating that foreign body. The paper in its entirety covers certain points most important about magnets. The giant magnet and the hand magnet are not interchangeable, nor is it permissible to consider the anterior and posterior routes as not having maximum optimum indications for use. The giant magnet is a magnet with a wide magnetic field, which can be well controlled and does not demand accurate foreign body localization. The British in their recent war surgery have demonstrated that to us. The hand magnet is a contact magnet and if one cannot approximate the foreign body with a minimum of 2 mm., there is no use whatever in trying even to use the hand magnet. The indications for anterior and posterior route extraction have been covered in the paper.

The Ophthalmologist's Place in the Prevention of Traffic Accidents. Lowell S. Selling, Detroit

With the development of visual standards for automobile drivers, the position of the ophthalmologist, as an appeal agent, becomes extremely important in the prevention of traffic accidents. He must evaluate how much ocular disease interferes with driving capacity. Refractive errors are probably less important than has been assumed in the past but there are a number of secondary considerations which must not be discounted in evaluating muscular weakness, monocolar vision and corneal pathology. Factors involved in glare sensitivity and night blindness must be carefully evaluated in each case. Common factors previously considered of great importance, such as color blindness and some types of diminished visual fields, can now be considered relatively insignificant. Cases which have passed through the traffic clinic in Detroit are cited as illustrations of these various points.

Discussion

Dr. S. Judd Beach, Portland, Maine: Dr. Selling's report tends to revolutionize our notions of the essential visual requirements for driving automobiles. Ophthalmologists are asked to recommend standards of vision for many occupations. Oddly they seem to feel no doubt that they are qualified to single out the exact line of the Snellen chart which determines the competence of a swimming teacher, aviator or automobile driver. One of the most painstaking and reasonable reports on requirements for automobile drivers has been submitted by the subcommittee of this section. Yet the members are doubtless aware that it is at best an attempt to translate monocular testing in the dim religious light of a refraction range into binocular driving on the road, and there is no correlation between the two. To illustrate, take the case of one of our bankers now 48 years old. With glasses his vision is better than 6/VI. Without them a high mixed astigmatism reduces his central acuity with both eyes open to 6/LX. Yet for years he has preferred to drive by day and to play golf and tennis without glasses, possibly because his vision taken in bright daylight with a contracted pupil is 6/XV, perfectly adequate for driving; more likely because he has become accustomed to going without glasses outdoors. The change in appearance of objects when glasses are worn presumably nullifies the improved central vision. A similar disturbance in space relations occurred in two fliers whom I examined this winter. Their slightly deficient central acuity obliged them to wear lenses while flying. Yet with these lenses their depth perception was definitely impaired. Analogous to this is aniseikonia. Lancaster tells of a driver who hogged the middle of the highway and carried

away the left doorpost of his garage until fitted with size difference lenses. It begins to seem in traffic accidents that many less obvious factors outweigh the customary criteria — visual acuity, fields and color perception. Factual studies such as these of Dr. Selling give us a sounder basis than the theoretic considerations previously followed.

Dr. Albert C. Snell, Rochester, N. Y.: Throughout the paper Dr. Selling uses the term "visual acuity" when reference is made to the Snellen acuity determinations and uses the term "vision" in its generic sense when he refers to the complete complex act of seeing. The punctilious and proper use of these terms is not generally followed by most American ophthalmologists, and this careless habit often causes confusion and misunderstanding. Dr. Selling points out that industrial capacity is not measured by acuity expressions. And I wish to add that neither do acuity expressions express an equivalent fractional ability of any visual function, not even of macular perception. Therefore one should not be surprised when Dr. Selling was able to come to the conclusion "Visual acuity does not, per se, evaluate the individual's ability to drive a car — visual acuity has nothing to do with ability to judge the speed and distance." Dr. Selling also finds that standard of 20/40 acuity is too rigid as the minimum required standard for safe driving. I derive satisfaction from the fact that Dr. Selling has been able to demonstrate this fact from his clinical observation of drivers. Evidently he has found that one is not one-half blind when visual acuity is reduced to 20/40, although some compensation boards still hold to the contrary view. When one considers the nature of the Snellen notation, a simple explanation is presented of Dr. Selling's observation that a person with 20/00 is able to drive efficiently and, therefore, that the Snellen acuity measurement does not measure the driver's aptitude. The ideal Snellen character, as we all know, is a five minute angle character each limb of which subtends one minute; therefore the normal eye can perceive easily a one minute object; since the 20/100 acuity angle is five times the 20/20, a five minute angle, an eye with a 20/100 acuity has an ability to detect an object equivalent in size to a one minute angle for the normal eye. The minimum visual acuity standards which have been established in several states are too rigid. Even those standards which were established by the committee of this section should be used as a guide and not as an ultimate fixed standard to be applied to each person applying for a license to drive an automobile. In my judgment, lay examiners should be instructed in more precise methods of screening in their visual testing. These should include, in addition to acuity testing, some simple methods of determining coordination of muscles. In the doubtful cases, at least, the final question of physical fitness for driving should be determined by the ophthalmologist in cooperation with an internist after a complete physical examination.

Dr. Lowell S. Selling, Detroit: My motive in presenting this paper was primarily to get expert opinion, and the two discussers, I feel, gave me an insight which I could not otherwise have as to the ophthalmologist's point of view. I have a secondary motive, for I am hoping that you will give further consideration to the elimination of vision testing by policemen and others who are not competent. Even such a simple procedure as that which Dr. Snell suggests, a simple test for muscular balance to be given by these license examiners, is impossible, as most lay examiners do not have enough intelligence quotient to be able to do such a test correctly. Then there is the problem of the optometrist who is passing out glasses without helping the traffic situation. If the Section on Ophthalmology can do anything to aid in correcting some of the false conceptions that have arisen in this field, it will be doing a great deal in behalf of safety.

Lymphomatous Tumors of the Eye and Its Adnexa (Lantern Demonstration). John S. McGavic, New York

A study of 23 cases of lymphomatous tumors involving the eye and its adnexa. In some cases the original lesion appeared in the region of the eye, while in others the eye was involved as a part of a previously diagnosed generalized lymphosarcomatosis, leukemia or lymphogranulomatosis (Hodgkin's disease). The classification of the lymphomatous processes is difficult because of varying opinions among pathologists. Clinical appearance, microscopic findings, treatment and prognosis. An attempt has been made to obtain a follow-up of each case. This is the most instructive part of the study, as it demon-

strates that all these tumors are ultimately more malignant than is usually thought. A localized tumor may regress completely after treatment only to have the same type of lesion appear at other sites after a latent period up to ten years. A localized lymphosarcoma of the iris is believed to be the first such case reported.

Ocular Changes in Young Diabetic Patients (Lantern Demonstration). Cecil S. O'Brien and J. H. Allen, Iowa City

Contrary to general belief, fundus changes occur in young diabetic patients probably as a result of the general disease. Punctate hemorrhages, cotton wool patches and occasionally hard, yellowish white degenerative areas similar to those observed in older diabetic patients have been seen in patients under 30 years of age. The hemorrhages and cotton wool patches were transitory. Transitory changes in refraction also were observed in young diabetic patients during periods of regulation. The occurrence of two varieties of diabetic cataracts has been reported previously. However, further observation has disclosed the development of lenticular opacities after prolonged periods of poor control of the diabetes. In some of these cases arrest of progress of the lens changes corresponded with the maintenance of adequate management of the general disease. Coincidental ocular changes were observed in a ratio similar to that for nondiabetic patients of the same ages.

Discussion

Dr. Glen Gregory Gibson, Philadelphia: Drs. O'Brien and Allen have again called attention to the ever increasing responsibility which ophthalmologists must assume in the field of internal medicine. In view of the relatively rapid progressive changes which are occurring today in the practice both of internal medicine and of ophthalmology, it becomes necessary that we assume an equally progressive point of view when the evidence is sufficiently convincing to warrant it. This paper is the result of the combination of progress in internal medicine in the form of the treatment of diabetes by insulin and careful clinical research in medical ophthalmology over a period of many years. The section is fortunate in having these authors bring to us the benefits of their extensive experience and studies in this most important subject. They stressed the increasing incidence of diabetic retinopathy in the younger persons, and I am in complete accord with their findings and explanation of these changes in the retinas of young persons with diabetes. It seems that this paper, in addition to many other excellent contributions by other authors, would once and for all put an end to that school of thought which denies the existence of a specific retinopathy that is pathognomonic of diabetes mellitus. Diabetic retinitis rather conveniently divides itself into a working classification of the well known central punctate type, the diffuse type, the mixed diabetic and hypertensive type and the venous type, any one of which may progress to retinitis proliferans and any one of which is usually sufficiently characteristic to permit an ophthalmoscopic diagnosis of diabetes. The case histories in this paper include examples of each of these types of retinopathy. It is well known that the conditions under discussion are not only resistant to treatment but, even under an adequate diabetic regimen as we know it today, are particularly prone to be progressive. Therefore it is encouraging to learn that in 6 of their 23 cases the retinopathy was transitory. This is consistent with the claims of our diabetic department that these retinal changes do not develop in the cases which are recognized sufficiently early and in which cooperation in the medical regimen is adequate. This would tend to increase our interest in demanding adequate medical management of these cases, particularly the early ones. The authors have also found evidence of the other well known ocular complication of diabetes in their younger patients, such as lipemic retinitis, hypotension, refractive changes and cataracts. Our experience has been similar with the exception of the incidence of cataracts in these young diabetic patients. In spite of careful search for these changes, we have been unable to detect them in any such frequency as they report in two series, namely 16 and 13.8 per cent. This paper has been of real value because it calls attention to the necessity of careful study of the younger as well as older diabetic patients and because it stresses the necessity of a rigid diabetic regimen for patients with this disease.

Ochronosis of the Sclera and Cornea Complicating Alkaptonuria: Review of the Literature and Report of Four Cases (Lantern Demonstration). James W. Smith, New York

A rare disorder of metabolism characterized clinically by excretion in the urine of homogentisic acid. In middle life pigmentation (ochronosis) is observed in cartilaginous, tendinous and fibrocartilaginous structures. An early clinical finding in ochronosis is blue discoloration of the auricular cartilages. Complete calcification of the intervertebral disks has been demonstrated recently by roentgenography before the clinical diagnosis of ochronosis was made. Eye findings were studied in four men aged 44, 54, 62 and 67. In the two younger men, brothers whose parents were first cousins, the only change noted was a faint brownish discoloration about the pingueculae. Prominent, discrete, slate gray areas situated deeply in the scleras temporally and nasally were seen in both eyes of the older patients. Many small brown pigment spots in the superficial layers of the corneas near the temporal and nasal limbi. The symmetrical corneal pigmentation represents a new biomicroscopic entity. Visual function was normal.

Discussion

Dr. M. L. Berliner, New York: In a case I examined the conjunctival pigmentation was seen in the form of ovular shaped brownish spots which under high power were composed of granules in a partially closed ring arrangement. The appearance and disposition of the pigment in the cornea is typical. It is found in Bowman's zone as small brownish discrete globules, resembling oil droplets symetrically arranged. It would be interesting to speculate on how this develops, i.e., whether the pigment migrates along the basal cell layers of the conjunctival epithelium into the cornea or whether it arises in situ. The fact that the pigment tends to develop in the nearly avascular tissues like tendons and cartilages might cause one to favor the latter idea.

Although alkaptonuria is a symptom of ochronosis it may occur as an isolated finding. Two types of ochronosis are known: first, the endogenous form, an inherited metabolic anomaly associated with alkaptonuria, and second, the exogenous form, resulting from phenol (carbolo-ochronosis) or benzine poisoning, including such substances as dinitrophenol, paradichlorobenzene, analine, adrenalin, and similar aromatic compounds. All these when ingested tend to form phenol derivatives in their intermediate metabolism. These intermediate products at the slightly alkaline **pH** of the body fluids polymerize into pigments. An analagous reaction occurs with adrenalin solution which when left exposed to air turns dark.

In the first or endogenous group (represented by Doctor Smith's cases), there is a total inability to destroy tyrosine and phenylalanine contained in the proteins of food and tissues, resulting in the excretion of homogentisic acid, an intermediary product. This substance is excreted into the urine. After many years due to the action of tyrosinase, there is a deposition of a black pigment similar to melanin in the cartilages and tendons.

I reported two cases of cataract from poisoning by inhalation of paradichlorobenzene vapor (a commonly used moth repellent). In one of these cases phenol derivatives were found in the urine. The urine turned dark on standing. Several months after the patients were removed from the action of the fumes they suddenly developed cataracts. In one case, a young woman of 30, the posterior cortex of the lens was definitely dark brown of the so-called nigra type. This was followed by a rapid swelling of the cortex. I would like to suggest that bio-chemical studies of tissues and excretions be made in such cases as black cataracts, melanosis oculi, whorl-like keratitis, Hudson's line, and others which are characterized by melanin or melanin-like pigmentation.

Dr. James W. Smith, New York: A pocket flashlight held behind the ear will delineate the opaque cartilage, affording a simple clinical diagnostic test. The ochronotic areas cannot be transilluminated.

I want to thank Dr. Berliner for his discussion. The ochronosis cases known definitely to be of alkaptonuric origin total 45. Of these 31 (69%) were males and 14 (31%) were females. This finding contrasts with statements in the literature that the sex incidence is about equal in men and women.

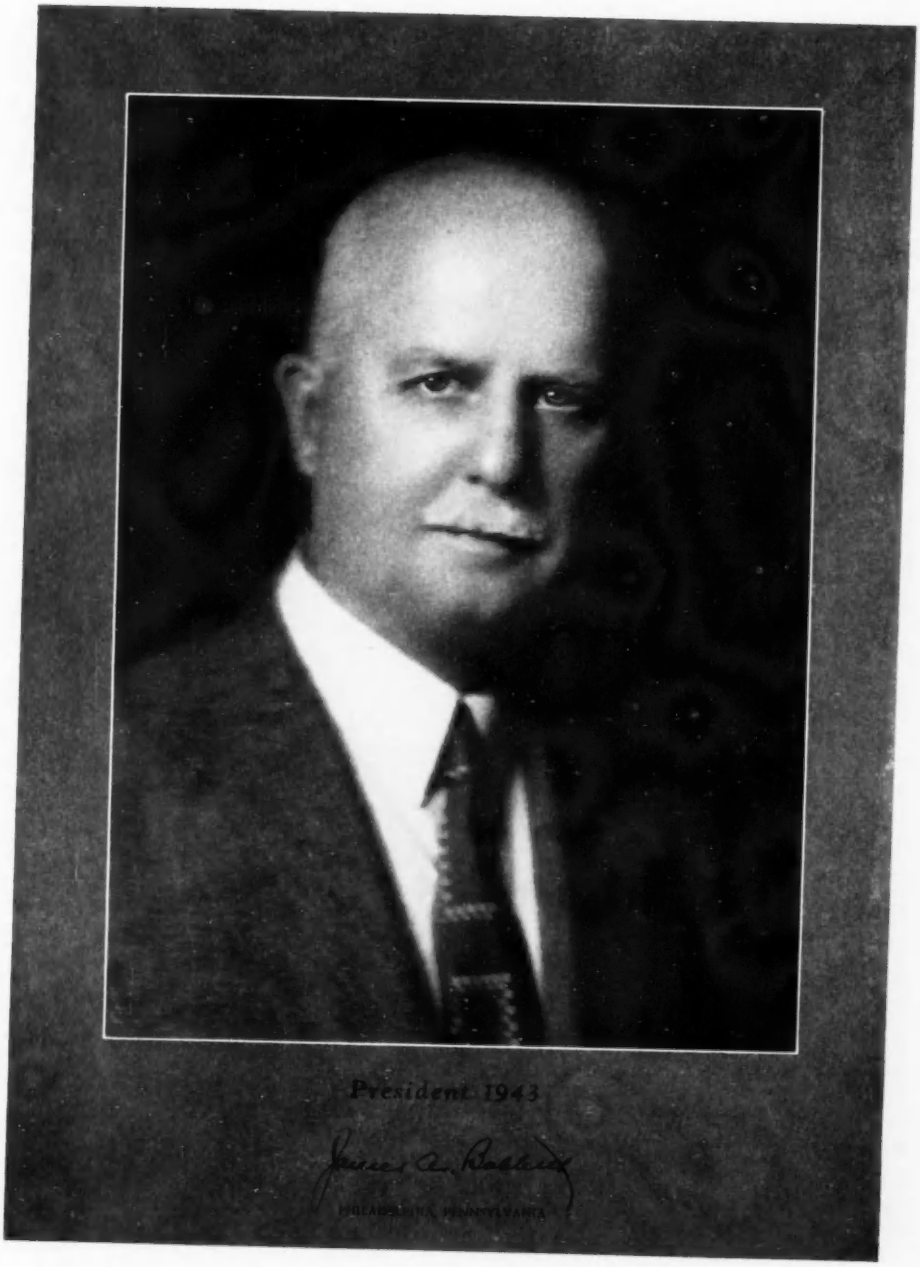
Many cases of alkaptonuria and ocular ochronosis were never recorded. In the

family of my second case alone there are five alkaptonurics and potential ochronotics not yet investigated.

It is important to remember that the eye picture is variable and progressive, starting as a faint, diffuse, brown discoloration in the palpebral fissure and ending as a slate colored and conspicuous brown-black scleral spot. I arranged my four cases chronologically in order to demonstrate this point.

The ophthalmologist should be able to aid the internist in detecting ochronosis by slit lamp examination in young adult patients. There is a possibility that some day homogentisic acid may be recovered from tears just as it can now be detected in urine.

There is no doubt in my mind that every alkaptonuric at birth is a potential case of ochronosis. By roentgenographic examination patients can be diagnosed in the second decade or possibly even sooner. Sealock, Galdston and Steele have suggested that the continued high intake of vitamin C in early life may prevent the deposition of melanotic pigment in later years.



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1935 — Wells P. Eagleton, M.D. ..... Newark, N. J.
1936 — Frank E. Burch, M.D. ..... St. Paul, Minn.
1937 — Lee W. Dean, M.D. ..... St. Louis, Mo.
1938 — Harry S. Gradle, M.D. ..... Chicago, Ill.
1939 — George M. Coates, M.D. ..... Philadelphia, Pa.
1940 — Frank R. Brawley, M.D. ..... Chicago, Ill.
1941 — Frank R. Spencer, M.D. ..... Boulder, Colo.
1942 — Ralph I. Lloyd, M.D. ..... Brooklyn, N. Y.

POSTOPERATIVE CICATRICIAL STRABISMUS
RESULTS OF THE TRANSPLANTATION OF TENON'S CAPSULE

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and
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BY INVITATION
NEW YORK

THE TECHNIC of transplanting Tenon's capsule for the correction of postoperative cicatricial strabismus caused by adhesions between the muscle and the sclera was published in 1938.[1] The results of 3 operations on 3 patients were described. Since that time 21 operations have been performed on 20 patients with postoperative squint, many of whom had already been unsuccessfully operated upon. Sixteen of these operations were successful. This operation has not corrected all the postoperative cicatricial deviations. But because several of our patients had had 2 or 3 operations to correct the deviation, which is usually an exotropia with almost complete limitation of adduction of the deviating eye, any operation which offers a chance for success seems worthy of trial.

In this paper new technics of operating as well as experiments performed on rabbits are discussed and clinical findings reviewed.

TECHNIC

In the original technic a piece of Tenon's capsule was excised, inverted and sutured over the roughened sclera beneath the muscle with chromic catgut sutures. In one case, Tenon's capsule was freed laterally and drawn toward the cornea; it was then sutured to the sclera to cover the roughened area. The muscle over the graft was resected.

With the present technic 2 sutures are placed at the edge of Tenon's capsule either superior or inferior to the muscle bed, depending upon the ease with which the capsule may be drawn into its new position. Two double-armed sutures are placed 10 mm. apart on the edge of the selected graft and Tenon's capsule is separated on either side of the sutures sufficiently to allow the graft to be drawn under the muscle without marked tension. In most of the recent cases the graft has not been reversed. The double-armed sutures are inserted beneath the episcleral tissue so that the site of previous adhesions of the muscle to the sclera are completely covered. If the graft does not lie smoothly in the new position other sutures may be inserted but the number of sutures should be minimum. An incision in Tenon's capsule parallel to the free border of the graft but at some distance from it may be required to permit the flap to slide freely.

In the majority of the more recent cases, nylon sutures (6-0)* have been employed entirely because it is felt that there is less reaction from these sutures than from those used previously. In several cases, a severe reaction had been noted after the graft had been su-

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Aided by a grant from The Ophthalmological Foundation, Inc.
Experimental studies performed in the Department of Research of the New York Eye and Ear Infirmary.
We are indebted to Dr. Daniel B. Kirby for permission to include several of his cases in this report.

* Made by Davis and Geck, Inc., Brooklyn, N. Y.

tured to the sclera with chromic catgut. Figures 1 through 7 show the technic of operation usually employed.

EXPERIMENTAL STUDY

Anatomy of Tenon's Capsule. — In order to understand thoroughly the results obtained, it is advisable to consider the anatomy of Tenon's capsule from a histologic as well as a gross anatomical standpoint. Fascia bulbi makes its appearance in the embryonic state at the 80 mm. stage as a sheet of mesodermal tissue developing in conjunction with the extra-ocular muscles.

Anatomically the tissue appears to be fused with the bulbar conjunctiva at the corneoscleral junction. It is possible by gross anatomical dissection to separate Tenon's capsule from the conjunctiva as far as the limbus. The tissue which histologically is a sheet of loose connective and fibrous tissue extends backward from the corneoscleral junction forming the capsules of the extra-ocular muscles and extending to the apex of the muscle cone where it appears to terminate. Tenon's capsule is divided into a thin inner or visceral layer and an outer layer or capsule proper.

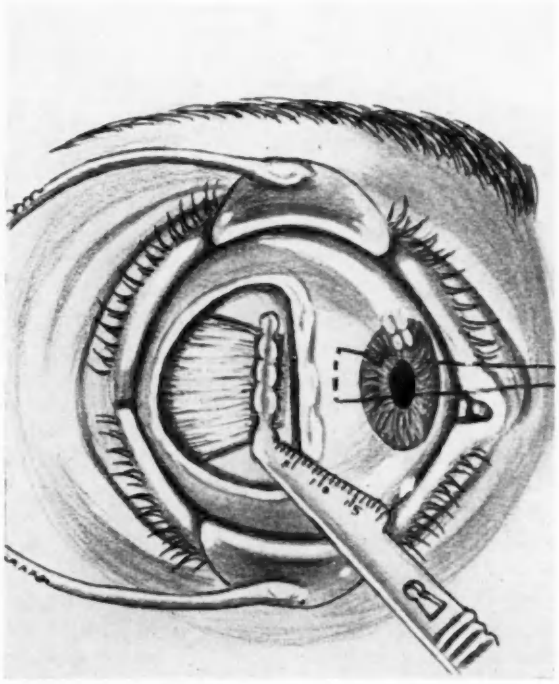


FIG. 1 — A vertical incision is made in the conjunctiva as close to the lateral canthus as possible, to expose the lateral rectus. Tenon's capsule is not disturbed over the muscle. One blade of a muscle forceps without teeth or pins is passed beneath the muscle at the tendinous insertion, or at the point desired for resection.

The study of Tenon's capsule from a histologic standpoint is very difficult because its fusion both with the overlying conjunctiva and the underlying scleral tissue is so marked that in his-

tologic preparations it is often almost impossible definitely to point out the tissue. On the other hand, clinically, it is usually easy to see the definite anatomic structure in its relations to overlying and underlying tissue. The explanation for this lies in the fact that in sectioning the eye the conjunctiva is matted down closely over the scleral tissue and no line of demarcation can be found showing the outline of the fascia bulbi. Gross dissection has so far proven the best means of study.

An attempt was made experimentally, using rabbits, to segregate the tissue by the injection of dyes between Tenon's capsule and its adjacent tissues. The dyes* were absorbed too easily and no satisfactory result could be obtained.

The anatomic and histologic study and its relation to surgical results explains the success of the surgical technic.

Rabbit Experiments. — In order to arrive at a definite conclusion in regard to the part Tenon's capsule transplant played in the restoration of function, the procedure was performed experimentally in rabbits.

Transplants were first made in normal rabbit eyes with as little trauma as

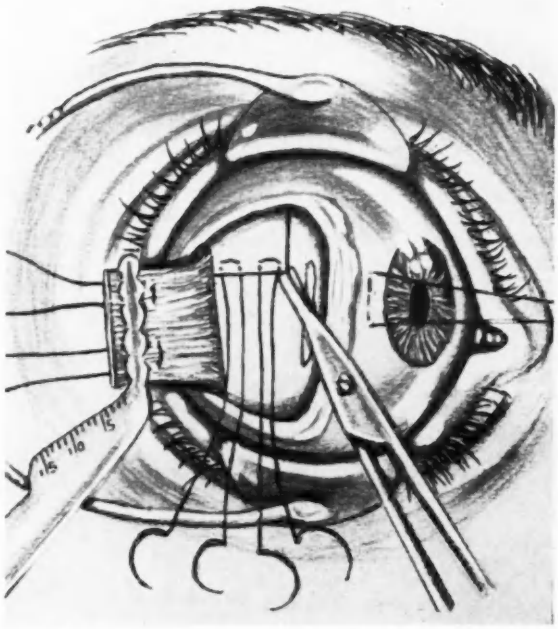


FIG. 2 — The lateral rectus is severed from its insertion, and the fibers lying between the forceps and the free border are excised. Two double-armed nylon sutures (6-0) are passed through the muscle from the scleral surface and then through channels in the forceps. Two double-armed nylon sutures (6-0) are passed through the border of Tenon's capsule at one side of the proposed graft. The incision in Tenon's capsule is indicated by a dark line.

* India ink, black and red, gentian violet, and methylene blue were used.

possible to the muscle and the surrounding tissues.

The operations were performed with sterile technic and at the conclusion of the operation either antiseptic collyria or ointment were used. The eyelids were sutured together at the termination of the operation. These sutures were allowed to remain in place from 24 to 48 hours and then were removed.

placed was exposed by an opening in the conjunctiva overlying the muscle followed by making a small opening in Tenon's capsule at one side of the muscle, and passing the tenotomy hook under the muscle. Two double-armed sutures were placed 2 mm. from the insertion and the muscle cut free and separated sufficiently to allow the placing of the Tenon's capsule transplant over

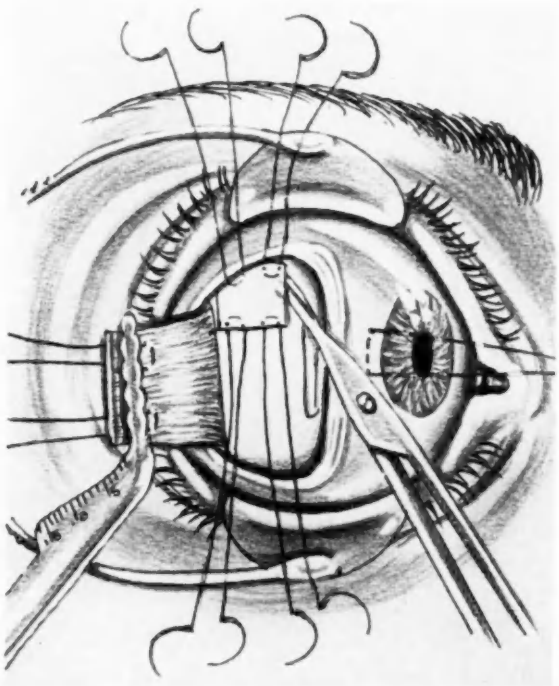


FIG. 3 — Two additional double-armed nylon (6-0) sutures are passed through Tenon's capsule opposite those previously inserted, and the graft is excised with Stevens' scissors.

Occasionally low grade conjunctival reactions occurred but only in three rabbits were the results discarded because of severe infection.

The superior and inferior recti muscles of rabbits were selected for experimental purposes because they were larger and apparently stronger than the lateral and medial recti muscles. The muscle under which the graft was to be

the muscle bed. After rotating the eye downward, the conjunctiva and Tenon's capsule on the opposite side of the eye were then infiltrated with saline in order to facilitate the separation of the fascia bulbi from the conjunctiva. The area overlying the muscle was avoided and the conjunctiva was separated from the underlying Tenon's capsule with Stevens' scissors until a sufficient-

ly large area of Tenon's capsule was exposed to permit transplantation.

With the tissue still in position, four double-armed sutures were placed at the corners of the proposed graft, the dimensions of which were made large enough to cover all rough areas on the sclera. Then using these sutures as a means of holding the tissue, Tenon's capsule was separated from the sclera and cut free keeping the graft in its normal position. The tissue was laid on the cornea or kept moist with physiologic saline solution to avoid its shriveling and in order to prevent curling of the tissue. The sutures were then placed in the episcleral tissue in such a manner that the graft would allow the muscle, when reattached to its insertion, to move freely over it without interference from the scleral sutures. When the sutures were placed at the desired points in the sclera, the graft was drawn down in position and the sutures tied. The double-armed sutures previously placed through the muscle were brought forward and replaced in the muscle stump and tied. The conjunctiva was approximated with continuous nylon sutures. The graft carried over in this manner was placed in some cases with its scleral surface in its normal position and in others the graft was reversed.

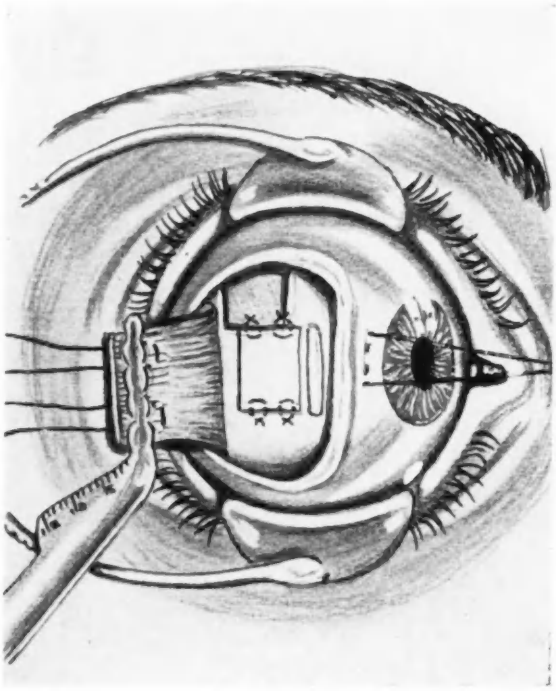


FIG. 4 — Tenon's capsule is reversed, and sutured with episcleral sutures over the rough scleral area where the lateral rectus was formerly adherent.

In the next method attempted a pedicle graft was used. The method was similar except that the graft was obtained from tissue in the vicinity of the muscle operated upon rather than from the opposite side of the eye.

After separating the muscle the edge of Tenon's capsule was grasped. Double-armed sutures were placed in its edges, approximately 10 mm. apart. A lateral incision was made on each side of the sutures to a point approximately 10 to 12 mm. away from the muscle bed. The transplant was then separated from the underlying sclera and was pulled over the muscle bed and sutured to the sclera; the muscle was sutured to its normal insertion. The graft was used in its normal position in some of the experiments and reversed in others.

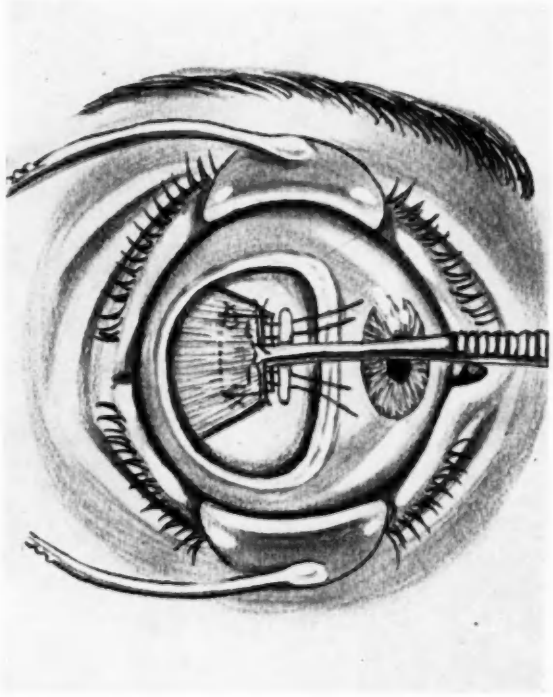


FIG. 5 — The lateral rectus is drawn over the graft by means of traction with fixation forceps, after passing the sutures through the muscle stump, or, the sutures are introduced to the corneal side of the insertion if advancement is indicated.

The last method used was performed without separating the muscle from its insertion. The conjunctival incision was the same as in previous experiments; the conjunctiva was separated from the underlying Tenon's capsule, the muscle edge was located at a point 3 to 4 mm. below the attachment and a small opening made in order to pass the muscle hook. The muscle was separated from the sclera and Tenon's capsule over a width of from 10 to 12 mm. Tenon's capsule was incised as far laterally as possible, frequently to a point nearly overlying the medial rectus muscle; a strip of Tenon's capsule 10 mm. wide was separated and carried downward toward the inferior rectus muscle to a point 2 to 3 mm. above the muscle. Two supporting sutures were placed through Tenon's capsule and the scleral tissue for fixation at this point. Two sutures were passed through the distal end of the graft. The graft was then reversed

on itself and passed under the intact inferior rectus muscle, carried under the muscle and back over the muscle and fastened at a point which approximated the two previously placed sutures. By this method a complete Tenon's apron graft surrounding the muscle was obtained (fig. 8).

Production of Adhesions in Rabbits' Eyes.—Although various methods were used to produce adhesions between the muscle and the underlying sclera as well as between the muscle and overlying conjunctiva, it is significant to note that by even slight manipulation of the muscle as would be made in the usual resection or recession, numerous adhesions were produced without the aid of other instruments or manipulations. Scarification of the sclera by means of a knife and abrasion of the muscle tissue with either a blunt or sharp instrument were used in the majority of cases to produce the desired adhesions. In some rabbits the adhesions were produced and the Tenon's transplant made at the same time using the above mentioned methods. In other rabbits the adhesions were produced and at a later date transplantation of Tenon's capsule was performed.

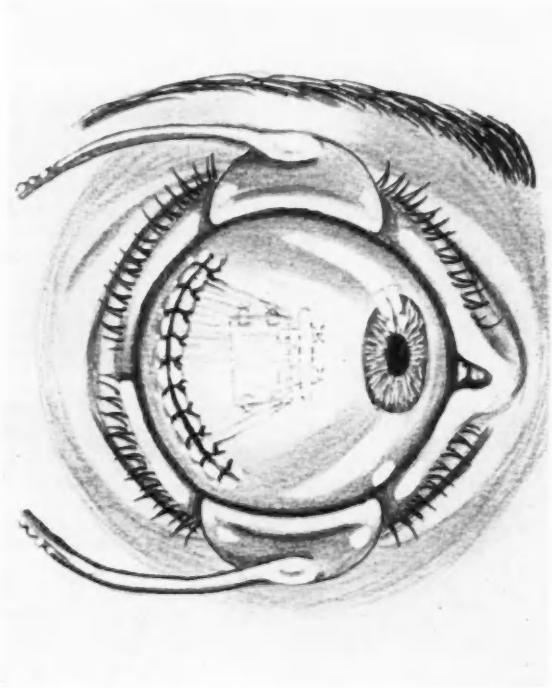


FIG. 6.—The conjunctival wound is closed with interrupted sutures of plain catgut (5-0). The muscle and the graft may be seen in outline through the conjunctiva.

Other Graft Materials.—It was considered advisable while studying the use of grafts of Tenon's capsule to use the other materials suggested as substitutes. Amnioplastin* was the first substitute used. This substance is a dried sterile amniotic membrane which is prepared after separation from the

* Obtained through the courtesy of Presbyterian Hospital and Dr. Algernon B. Reese.

placenta at the time of delivery. It has been believed that this substance prevents adhesions because it does not fuse with surrounding tissue. The tissue proved unsatisfactory in our experiments because: (1) It was extremely friable and the sutures pulled through preventing satisfactory fixation of the transplant in its new position overlying the muscle bed; (2) the tissue was difficult to handle from a technical standpoint and it was constantly rolling itself into a ball in such a manner that it was almost impossible to smooth it out in the correct position, and (3) when the graft was placed satisfactorily at the time of operation, no effective result was noted postoperatively.

Egg membrane was the second substance used and this proved to be unsatisfactory in most cases because it acted more as a foreign body than as a graft tissue.

Peritoneum was tried and discarded for the same reasons as amnioplastin.

The experimental data on the transplantation of Tenon's capsule and other materials on rabbits are tabulated in table I.

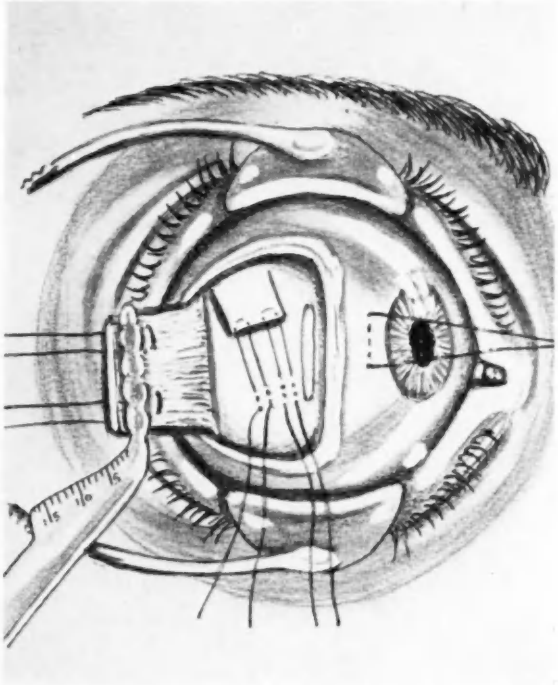


FIG. 7 — Technic for using a pedicle flap of Tenon's capsule. Note how nylon sutures are placed on the other side of the scarred episcleral area.

Discussion of Table I. — From table I it may be seen that post-operatively in every rabbit there were some adhesions between the muscle and the underlying sclera as well as between Tenon's capsule and its overlying muscle and underlying sclera. Evidence points to the fact that the scleral surface of Tenon's capsule in the majority of rabbits is less likely to form strong bands of ad-

TABLE I

Results of Transplanting Tenon's Capsule and Other Substances on 47 Rabbits' Eyes

Number of Rabbits	*Description of Graft*	*Type of Trauma*	*Adhesions*	*Extent of Adhesions*	*Adhesions after Secondary Tenon's Capsule Transplant*	*Results after Tenon's Capsule Transplant*	*Remarks*
3	Free Tenon's capsule—unturned	None	Firm to muscle; few to sclera	15 mm. from muscle insertion.		Adequate motion of muscle.	Adhesions appear insufficient to reduce motility of muscle.
3	Free Tenon's capsule—reversed	None	Firm to sclera; few to muscle	10 to 12 mm. from attachment of muscle.		Moderately good motility.	Adhesions apparently limit motion less than in group of experiments in which Tenon's capsule was not reversed.
4	Pedicle of Tenon's capsule pulled down; unturned	None	Firm to muscle; few to sclera	To the equator		Good motility.	Apparently better results when transplant not fixed at four points.
4	Pedicle of Tenon's capsule pulled down and reversed	None	Firm to sclera; few to muscle	10 to 12 mm. from muscle insertion.		Good motion.	Somewhat better results when graft turned on itself.
4	Apron	None	Moderate to sclera and muscle	8 to 10 mm. from muscle insertion.		Good motility.	Apron appears to allow the greatest motility of the muscle.
2	Amnioplastin	None	Firm to sclera and muscle	Past equator and to surrounding tissue.		Unsatisfactory	Considerable foreign body reaction.
1	Egg membrane	None	Firm to sclera and muscle	To equator		Unsatisfactory	Complete fixation. Results unsatisfactory.

TABLE I — *Continued*

Results of Transplanting Tenon's Capsule and Other Substances on 47 Rabbits' Eyes

Number of Rabbits	*Description of Graft*	*Type of Trauma*	*Adhesions*	*Extent of Adhesions*	*Adhesions after Secondary Tenon's Capsule Transplant*	*Results after Tenon's Capsule Transplant*	*Remarks*
1	Egg membrane; side reversed	None	Firm to sclera and muscle	To equator		Unsatisfactory	Complete fixation. Results unsatisfactory.
2	Peritoneum	None	Moderately firm	10 to 12 mm. from attachment of muscle.		Slight muscle action.	Tissue not satisfactory.
1	Free unturned	Scarification of sclera and trauma to muscle	Relatively firm; to muscle and sclera	Well beyond equator.		Comparatively good motility of muscle.	Despite adhesions a fair amount of action was left.
2	Free unturned	Scarification of sclera and muscle	Muscle firmly fixed to sclera and conjunctiva after first operation	Muscle firmly fixed well beyond sclera.	Less marked	The fixation which resulted from original trauma reduced markedly by Tenon's transplant; fair motility.	Although some adhesions present after implant of Tenon's capsule, motility of muscle increases from 0 to 60 per cent.
1	Free reversed	Scarification of sclera and muscle	Firm adhesions to sclera and muscle	To the equator		Good motility.	Action of muscle fair despite adhesions.
2	Free reversed	Scarification of sclera and muscle	Completely fixed to sclera and muscle	Past the equator	Motility increased to better than 50 per cent.	Firm fixation reduced.	Tenon's capsule transforms a fixed muscle into one with better than 50 per cent action.

TABLE I — *Continued*

Results of Transplanting Tenon's Capsule and Other Substances on 47 Rabbits' Eyes

Number of Rabbits	*Description of Graft*	*Type of Trauma*	*Adhesions*	*Extent of Adhesions*	*Adhesions after Secondary Tenon's Capsule Transplant*	*Results after Tenon's Capsule Transplant*	*Remarks*
1	Pedicle unturned	Scarification of sclera and muscle	Firm fixation to muscle and moderately firm to sclera	To the equator		Motility fair.	Apparently better result than with free graft.
2	Pedicle unturned	Scarification of sclera and muscle	Firmly adherent to muscle and sclera	Well past equator	Results improved considerably by transplant.	Complete fixation relieved.	Results improved considerably by transplant.
2	Pedicle reversed	Scarification of sclera and muscle	Moderately fixed to muscle and sclera	To the equator		Motility better with the reversed graft than with the unreversed.	Results show better motility than when unturned.
2	Pedicle reversed	Scarification of sclera and muscle	Complete fixation to muscle and sclera.	Well past equator.	Tenon's transplant increases the motility of the muscle considerably.	Fixation complete until secondary Tenon's capsule transplant.	Results in these cases were better than in the free graft.
2	Apron	Scarification of sclera and muscle	Moderate adhesions to muscle; more marked to sclera.	To the equator	Moderate amount of adhesions.	Good motion.	Action was better than in other methods.
6	Apron	Scarification of sclera and muscle	Completely bound to sclera and muscle.	Well beyond equator.	Moderate adhesions to sclera; less marked to muscle.	Muscle action practically entirely restored in areas of graft.	Seems to be the most satisfactory method used experimentally for increasing muscle function.

hesion than the conjunctival surface. On the other hand, in the presence of trauma it has been shown definitely that firm bands are formed to both the sclera and the muscle. Even the trauma encountered in manipulation of a muscle for resection or recession is often sufficient to cause adhesions between the muscle and the sclera.

The experiments in which sliding grafts were used produced better results probably because the graft was less firmly fixed in its final position and there was less reaction from the smaller number of sutures.

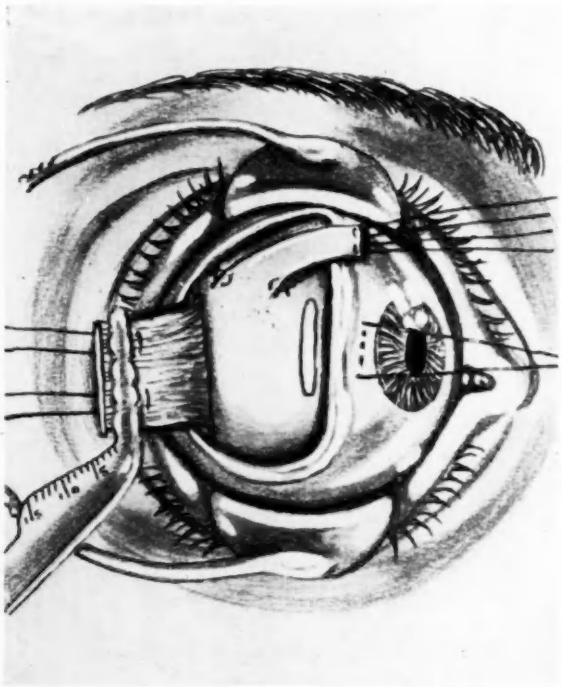


FIG. 8 — Preparation of apron graft.

The apron graft from a mechanical standpoint apparently forms a smooth tunnel in which the muscle may slide easily. In any rabbit in which a Tenon's capsule transplant was used there was never complete freedom from adhesions and, therefore, our explanation of the results must refer back, as mentioned previously, to the anatomy. Tenon's capsule, as stated before, is a loose connective and fibrous tissue. The fact that the tissue is so constructed is apparently responsible for the results, and even if there are adhesions to the anterior and posterior surfaces the capsular tissue still permits a moderate amount of play between the muscle and the sclera. It would seem, therefore, that Tenon's capsule not only prevents to some extent adhesions from forming between the sclera and the muscle, but also because of the structure of the capsule allows some motion of a muscle that previously had been bound down and immobilized by firm fibrous bands.

In the microscopic study of the postoperative condition in rabbits it was found that there was invariably a moderate amount of adhesions. When the

results were unsatisfactory it was more difficult to see the lines of demarcation between Tenon's capsule, the muscle and the sclera than under normal conditions. Therefore, after studying six rabbits' eyes the histologic method of study was rejected as unsatisfactory and gross dissection was performed on the remaining eyes.

a bad result in postoperative cicatricial exotropia; case 3 a good result in cicatricial exotropia following a second transplantation of Tenon's capsule in a patient whose cosmetic appearance and functional result were worse after the first transplantation of Tenon's capsule; case 4 a good result in postoperative cicatricial esotropia.

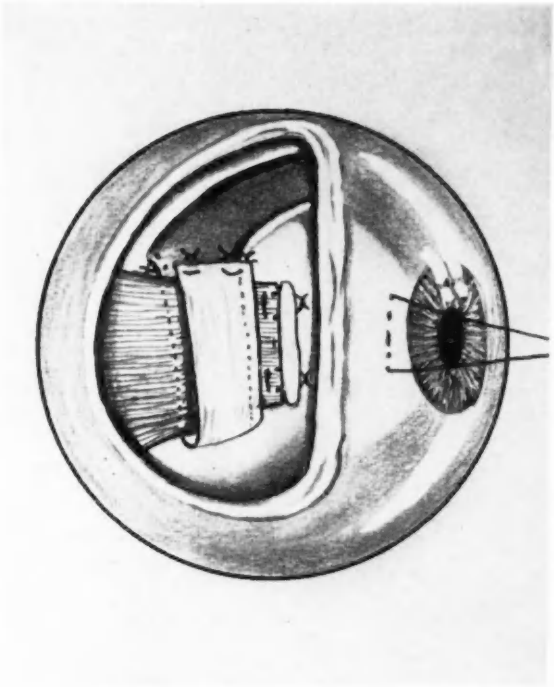


FIG. 9 — Apron graft of Tenon's capsule passed under the muscle, then over it where it is sutured in position.

CASE REPORTS AND RESULTS OF TWENTY-ONE OPERATIONS PERFORMED ON TWENTY PATIENTS

The case reports* are so extensive that only four somewhat typical cases have been selected for presentation. Case 1 records a good result in postoperative cicatricial exotropia; case 2

Case 1. — Postoperative cicatricial exotropia improved by reversed pedicle flap of Tenon's capsule: R. K., 13 years.

Operations performed prior to transplantation of Tenon's capsule:

Recession of medial rectus.

Resection of lateral rectus for convergent strabismus, O.S., 6/3/32.

Preoperative examination — January, 1942:

Visual acuity without correction:

O.D. 20/30-3

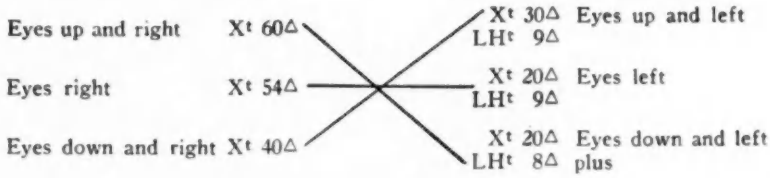
O.S. 20/70

* Key to abbreviation used in case reports: O.D., right eye; O. S., left eye; Xt, exotropia; LHt, left hypertropia, RHt, right hypertropia; NPc, near point of convergence; Et, esotropia.

Postcycloplegic acceptance:
O.D.+6.00 sph.⊃+1.00 cyl., axis 180°—20/30
O.S.+6.00 sph.⊃+1.00 cyl., axis 180°—20/70

Muscle balance: Screen test with prisms — without correction: At 6 M. Xᵗ 40Δ
LHᵗ 9Δ
At 25 cm. Xᵗ 38Δ
LHᵗ 9Δ

Cardinal Positions of Gaze (screen test with prisms, 1 M. distance):



NPc: No relative convergence. Left eye remains in divergent position.

Cover Test: Fixes with O. D.

Motility: Postoperative cicatricial or insertional paresis of left medial rectus — secondary contracture of the left lateral rectus (direct antagonist). Underaction of left inferior rectus — secondary contracture of left superior rectus.

Fields of Monocular Fixation: Limitation of adduction 23°, O. S.

Correspondence: Major amblyoscope (synoptophore) — anomalous.

Objective angle:	Subjective angle:
Xᵗ 24Δ	Eᵗ 10Δ
LHᵗ 5Δ	LHᵗ 5Δ

Surgery: Advancement and resection of medial rectus with Tenon's capsule transplant, O. S. — 4/10/42. The conjunctiva was incised over the left medial rectus. Tenon's capsule was incised and the muscle exposed on a hook. A resection clamp was placed, the muscle cut at its insertion, and double-armed nylon resection sutures placed and a 6 mm. resection performed. The old adhesions between the muscle and the sclera were freed as far back as the equator. The conjunctiva was dissected free of Tenon's capsule above the muscle and a strip of Tenon's capsule exposed. Double-armed nylon sutures were placed at the edges of the exposed strip and a flap dissected free except for a small pedicle. The transplant was reversed and pulled inferiorly so that it covered the area where the adhesions were cut. It was then sutured to the sclera in this position. The muscle was then brought forward over Tenon's capsule and reattached to the original point of insertion of the muscle.

Postoperative Examination — July, 1942:

Visual acuity without correction:
O. D. 20/40
O. S. 20/50

Refraction: Same as before operation.

Muscle balance: Screen test with prisms — without correction:

At 6 M.	At 25 cm.
Xᵗ 8Δ	Xᵗ 15Δ
LHᵗ 5Δ	LHᵗ variable

Cardinal Positions of Gaze (screen test with prisms — 1 M. distance): Deviation fairly comitant; slight increase in eyes right (lateral deviation); vertical same as before operation, increasing eyes down and left (weakness of left inferior rectus — secondary contracture of left superior rectus).

Eyes right	Eyes left
Xᵗ 23Δ	Xᵗ 6Δ
LHᵗ 2Δ	LHᵗ 2Δ

NPc: 90 mm.

Motility: Adduction, O. S. excellent.

Fields of Monocular Fixation: Left eye — adduction 49°, abduction 45°.

Correspondence: Major amblyoscope (synoptophore):

Objective angle X^t 10△; with massage could
LH^t 2△
superimpose.

Recommendation: Converging exercises after development of normal correspondence.

Result: Excellent cosmetic result and a good restoration of motility in the field of action of the adherent medial rectus. Normal projection developed; convergence restored with binocular single vision with eyes in primary position.

Case 2. — Postoperative cicatricial exohypertropia not benefited by advancement prior to the transplantation of Tenon's capsule and condition worse after the transplant: P. A. B., 16 years. Operated upon through the courtesy of Dr. L. W. Gorton.

Operations performed prior to transplantation of Tenon's capsule: Advancement of the lateral rectus and recession of the medial rectus, O. S., June, 1931; advancement of medial rectus, O. S., September, 1931.

Preoperative Examination — October, 1938:

Visual acuity without correction:

O. D. 20/15
O. S. 20/20

Postcycloplegic acceptance: (not prescribed).

O. D. +0.25 sph.
O. S. +1.00 sph. ⊃+0.50 cyl., axis 110°

Muscle balance: Screen and parallax test:

At 6 M. X^t 35△ LH^t 10△ At 25 cm. X^t 50△ LH^t 10△

NPc: Relative NPc remote.

Motility: Marked limitation of adduction, O. S.

Fields of Monocular Fixation: Limitation of adduction 30°, left eye.

Correspondence: Major amblyoscope (orthoptoscope) — Normal objective angle:

X^t 25△; Amplitude: 35△ base in,
LH^t 5△
40△ base out; third grade fusion.

Surgery: Resection of the left medial rectus, advancement and transplantation of Tenon's capsule and retroplacement of the lateral rectus 10 mm. from cornea, O. S., 10/15/38. The medial rectus of the left eye was exposed and adhesions were found extending 20 mm. from the cornea between the muscle and the sclera. A 10 mm. piece of Tenon's capsule was excised from the upper border of the capsule and brought down and placed over the adhesion without turning it over because the scleral surface of Tenon's capsule was congested and rough. Tenon's capsule was sutured in place with chromic catgut sutures and the medial rectus advanced to 4.5 mm. from the cornea with 10 day 0000 chromic catgut sutures. A larger adhesion was found extending from the lower border of the muscle at about 15 mm. from the cornea, probably accounting for the hypotropia. The left lateral rectus was receded with chromic catgut sutures to 10 mm. from the cornea and the eye seemed to be straight and did not deviate upward after the operation. Numerous adhesions were found under the left lateral rectus. The conjunctiva was closed with running catgut sutures.

Postoperative examination — June, 1939:

Visual acuity without correction:

O. D. 20/15
O. S. 20/70 PH 20/25+1

Muscle balance: Screen and parallax:

At 6 M. X^t 65△ LH^t 4△ At 25 cm. X^t 75△ LH^t 6△

NPc: Unimproved.

Motility: Marked limitation of adduction, O. S. Paresis of right superior rectus, secondary overaction of left inferior oblique.

Fields of Monocular Fixation: Limitation of adduction, 35°, left eye.

Correspondence: Major amblyoscope (orthoptoscope)—normal; same as preoperative findings.

Remarks: Cosmetic and functional results were worse and deviation greater. The reaction to the operation was great and was presumably caused by the buried chromic catgut sutures, which have since been discarded for nylon sutures. Probably more adhesions formed between the muscle and the sclera.

Case 3. — Postoperative cicatricial exotropia made worse by the first transplantation of Tenon's capsule but improved by a second transplant: J. H., 9 years.

Operations performed prior to transplantation of Tenon's capsule: Bilateral retroplacement of the medial recti, 3 mm. 7/15/35; bilateral myomectomy of the inferior obliques, 7/13/39.

Preoperative examination — October, 1939:

Visual acuity without correction:

O. D. 20/25+1
O. S. 20/20

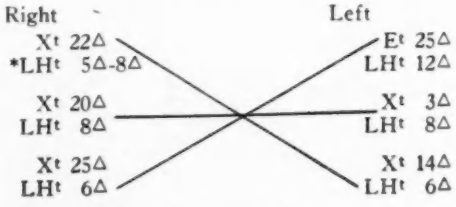
Postcycloplegic acceptance:

O.D.+0.50 cyl., axis 95°—20/20; 2Δ base up
O.S.+0.37 cyl., axis 85°—20/20-2; 3Δ base down

Muscle balance: Screen and parallax:

At 6 M. X^t 15Δ Double — LH^t 20Δ Hypertropia
At 25 cm. X^t 7Δ — LH^t 10Δ

Cardinal positions of gaze:



NPc: 50 mm. relative.

* Double hypertropia.

Motility: Marked limitation of the left medial rectus; paresis of right and left inferior oblique; overaction of the right and left superior rectus and right medial rectus.

Fields of Monocular Fixation: 15° limitation, O. S.

Correspondence: Normal.

Objective angle X^t 10Δ; superimposition. LH^t 4Δ

Surgery: Tenon's capsule transplant under the left medial rectus with a 5 mm. resection of the muscle, O. S.; central partial tenotomy of the left superior rectus, November, 1939. Under ether anesthesia, an incision was made over the left superior rectus. The muscle was then isolated and a tenotomy of the center two-thirds was performed. The conjunctiva was closed with running catgut. The left medial rectus was exposed and isolated and the point of attachment from which pull was exerted because of adhesions was found to be 11 mm. from the limbus. There was also one adhesion at the lower border of the muscle which did not appear to be very strong. A piece of Tenon's capsule about 7 mm. square was excised and sutured over the former area of attachment of the medial rectus and adhesions from this muscle. A 5 mm. resection of the medial rectus was then performed and it was reattached 5.5 mm. from the limbus. The conjunctiva was closed with running catgut suture. A pressure dressing was applied. Hole in mask before eye not operated upon.

Immediate postoperative examination — November, 1939:

Visual acuity without correction:

O. D. 20/20-
O. S. 20/20-

Muscle balance without correction:

At 6 M. X^t 14Δ — LH^t 9Δ
At 25 cm. X^t 20Δ — LH^t 5Δ

Correspondence: Major amblyoscope (orthoptoscope)—without correction—normal.

Xt 5Δ
LHt adjustment
Extorsion of left image.

Recommendation: Amplitude training.

Preoperative examination — September, 1940:

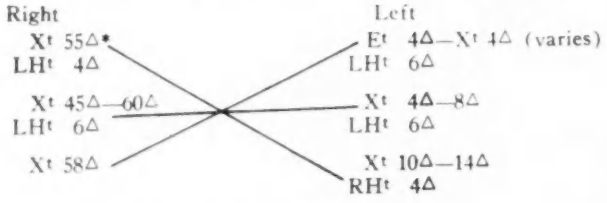
Visual acuity without correction: same as before.

Muscle balance without correction: screen test —

At. 6 M. Xt 45Δ RHt 6Δ Double hypertropia
At 25 cm. Xt 56Δ LHt 4Δ Intorsion, O.S.

Motility: Marked limitation of adduction (33°) of left eye.

Cardinal positions of gaze:



NPc: 45 mm. relative, O. S.

* Double hypertropia.

Surgery: After consulting with Doctor Kirby and Doctor White it was decided to perform another transplantation of Tenon's capsule in spite of the bad result from the first operation. Tenon's capsule transplant, O. S. with resection of medial rectus, which was adherent to the sclera 18 mm. from the cornea, 9/11/40: The muscle was located and clamped, and cut from its insertion, resecting it 7 mm. Two double-armed silk sutures were placed in the muscle. A 7 mm. square of Tenon's capsule was taken from the upper nasal quadrant. This was not inverted because of the roughness of the under surface. It was then sutured in place at its four corners with white silk (8-0) under the the muscle over the roughened sclera. The medial rectus was then sutured to the sclera 5 mm. from the limbus. A running continuous plain catgut suture (5-0) was used to close the conjunctival wound.

Postoperative examination —

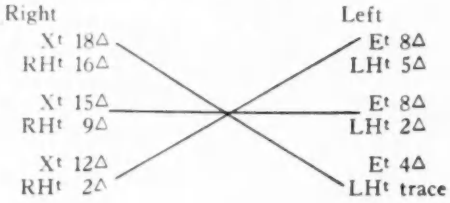
Visual acuity without correction:

O. D. 20/20
O. S. 20/20

Muscle balance: Screen test —

At 6 M. Et 8Δ RHt 7Δ LHt trace
At 25 cm. Et 6Δ RHt 7Δ LHt trace

Cardinal positions of gaze:



NPc: 45 mm. relative, O. S.

Cover test: Periodic phoria.

Motility: Slight limitation of adduction of the left eye. Overaction of the superior recti secondary to paresis of the inferior oblique (postoperative); fairly marked intorsion of left eye.

Fields of monocular fixation: Limitation of adduction, O. S. (15°).

Correspondence: Major amblyoscope (synoptophore) —

Objective angle: Et 12Δ
RHt 5Δ-8Δ
Torsion: adjustment.

Second grade binocular vision.
Amplitude: 25Δ base out to 4Δ base in.

Recommendation: Diverging exercises.

Remarks: Excellent cosmetic and good functional result after the second

transplantation of Tenon's capsule. After the first transplantation of Tenon's capsule, the cosmetic appearance was worse and the functional impairment was greater than before operation.

Case 4. — Postoperative cicatricial esotropia: E. F., 7½ years (data on operations and examinations, courtesy of Dr. D. B. Kirby).

Operations performed prior to transplantation of Tenon's Capsule: Bilateral resection of the lateral recti, April, 1938; bilateral recession of the medial recti, August, 1938.

Preoperative examination —

Visual acuity with correction:

O. D. 20/40-
O. S. 20/30

Postcycloplegic acceptance:

O.D. +2.00sph. ⊂+3.00 axis 90°
O.S. +3.75sph. ⊂+2.00 axis 60°

Muscle balance without correction:

At 6 M. Et 50Δ At 25 cm. Et 65Δ

Motility: Limitation of abduction of both eyes. No diplopia; alternate suppression — no fusion.

Surgery (Dr. Kirby): Resection and recession, O. D., with Tenon's capsule transplant, 5/13/41. The right eye was prepared in the routine manner. Argyrol and adrenalin were instilled. The speculum was inserted. The conjunctiva was incised over the insertion of the externus. The muscle was delivered on a hook and freed of its fascial attachments. Many adhesions were encountered. The muscle was grasped with a Reese clamp and sectioned from the eyeball. Tenon's capsule transplant was prepared over the bed of the muscle. By using two single-armed white silk sutures, a 6 mm. resection was performed. The conjunctiva was closed with interrupted plain gut sutures. The conjunctiva was then incised over the insertion of the internus. The muscle was delivered on a hook and freed of its fascial attachments. By using two single-armed white silk sutures, a 4 mm. recession was performed. The conjunctiva was closed with interrupted plain catgut sutures. A monocular dressing was applied.

Postoperative examination — December, 1941:

Visual acuity: with correction:

O. D. 20/20-
O. S. 20/20-

Refraction:

O.D.+1.75 sph. ⊂+2.50 cyl., axis 85°
O.S.+3.50 sph. ⊂+1.00 cyl., axis 80°

Muscle Balance:

At 6 M. Xt 3½Δ At 25 cm. Xt 4Δ
LHt 5Δ

Motility: No diplopia; alternate suppression; second degree fusion at times; good rotation; eyes straight.

Result: Excellent cosmetic result and a good functional result.

Statistical Results — Data on the entire group of patients are tabulated in tables II, III, IV, V and VI. In 16 of the 21 operations there was definite improvement of convergence following operation. The limitation of motion, which is usually the most serious indication for undertaking this procedure, was improved in the majority of cases. In three cases there was insufficient data, in two cases limitation was increased, and in the remaining 16 cases limitation was partially or completely corrected. The average improvement was 50 per cent. Practically complete restoration of function was obtained in 4 cases. In one of these cases the first transplantation of Tenon's capsule made the cosmetic appearance and function worse, but the second operation almost completely restored adduction, and the cosmetic result was excellent.

The postoperative deviation showed average improvement of 50 per cent in the exotropic cases and a 66 per cent improvement in the esotropic cases. The vertical imbalance was also definitely improved.

TABLE II*

Transplantation of Tenon's Capsule — Preoperative Findings and Postoperative Results

No.	Preoperative Findings				Postoperative Results				Cosmetic Result	Functional Result
	Deviation		Adduction	Convergence	Deviation		Adduction	Convergence		
	6 M.	25 cm.			6 M.	25 cm.				
1	Xt40△ LHt9△	Xt38△ LHt9△	23° limitation	None	Xt8△ LHt5△	Xt15△ LHt 2△	No limitation	Remote; but improved	Excellent	Good
2	Xt40△ LHt35△	Xt28△ LHt35△	Marked limitation	Remote	0	Xt16△ LHt 8△	Adduction limited	Improved	Excellent	Good
3	Et17△ LHt8△	Et17△ LHt6△	10° limitation	Remote	Xt3△	Xt 10△	Poor	80 mm.	Good	Fair
4	Xt47△	Xt57△	Limited	Remote	Xt50△	Xt62△	No limitation	50 mm.	Poor	Good
5	Xt18△ RHt5△	Et trace RHt5△	Limited	Remote	Xt8△	Xt1△—2△	None	130 mm.	Excellent	Good
6	Xt and Et variable	Xt and Et variable	33° limitation	130 mm.	Et10△	Et24△	10° limitation	85 mm.	Good	Good
7	Xt46△ LHt8△	Xt63△ LHt8△	Complete limitation	None	Xt29△	Xt38△	20° limitation	Remote	Fair	Fair
8	Xt18△ RHt2△	Xt19△ RHt2△	45° limitation	Remote	Xt12△	Xt9△ RHt4△	Marked limitation	Improved	Poor	Poor
9	Xt40△	—	—	—	Xt 6△ RHt2△	Xt 7△ RHt2△	Limited	130 mm.	Good	Fair
10	Xt84△	Xt84△	40° limitation	Remote	Xt3△	Xt4△	22° limitation	None	Excellent	Fair
11	Xt35△	Xt45△	—	Remote	Xt6△	Xt13△	—	90 mm.	Good	Fair

* Key to abbreviations: Xt, exotropia; Et, esotropia; LHt, left hypertropia; RHt, right hypertropia; O, orthophoria; —, no data.

TABLE II — *Continued*

Transplantation of Tenon's Capsule — Preoperative Findings and Postoperative Results

No.	*Preoperative Findings*				*Postoperative Results*				*Cosmetic Result*	*Functional Result*
	Deviation		*Adduction*	*Convergence*	*Deviation*		*Adduction*	*Convergence*		
	6 M.	25 cm.			6 M.	25 cm.				
12	X†30△	X†35△	Weak	Remote	E†13△	E†20△	Good	Improved	Good	Good
13	E†45△ LH†12△	E†50△ LH†15△	Limited	120 mm.	X†23△	X†35△	Markedly improved	Improved	Fair	Fair
14	E†50△	E†65△	—	—	X† 3.5△	X†4△	—	—	Excellent	Good
15	X†25△	X†30△	Marked	250 mm.	X†55△	X†60△	Adduction improved	Improved	Bad	Poor
16 (1)*	X†15△ LH†20△	X†7△ LH†10△	Limited	50 mm. relative	X†14△ LH†9△	X†20△ LH†5△	—	—	Bad	Bad
(2)	X†14△ RH†6△	X†56△ RH†4△	—	—	E†8△ RH†7△	E†6△ RH†7△	Slight limitation	45 mm.	Excellent	Good
17	X†49△ RH†12△	X†54△ RH†8△	29° limitation	None	X†23△ RH†8△	X†20△ RH†8△	7° limitation	70 mm.	Good	Excellent
18	X†55△ RH†12△	X†55△ RH†12△	40° limitation	300 mm.	X† 24△	X†45△	17° limitation	260 mm.	Good	Good
19	X†35△ LH†10△	X†50△ RH†10△	30° limitation	Remote	X†65△	X†75△	35° limitation	Remote	Bad	Bad
20	X†7△ RH†4△	X†8△ RH†4△	30° limitation	—	X†18△	X†20△	20° limitation	—	Poor	Fair

* First transplantation.

TABLE III

Average Preoperative and Postoperative Deviation

Number of Cases	*Preoperative*	6 M.	25 cm.	*Postoperative*	6 M.	25 cm.
19	Exotropia	33Δ	34Δ	Exotropia	15Δ	19Δ
21	Hypertropia	6.5Δ	6Δ	Hypertropia	2Δ	1.5Δ
2	Esotropia	48Δ	58Δ	Esotropia	13Δ	20Δ

TABLE IV

Average Limitation of the Field of Monocular Fixation

No. of Operations	*Pre-operative*	*Postoperative*
8	34Δ	10Δ
3	No data	No data
2		Unimproved or slightly improved
3		Moderate improvement
5		Good improvement
21	— Total	

TABLE V

Postoperative Convergence

	No. of Operations
Insufficient data	3
Unimproved	2
Moderate improvement	7
Marked improvement	9
Total	21

TABLE VI

Cosmetic and Functional Results

	Cosmetic	*Functional*
Excellent	6	1
Good	7	9
Fair	2	7
Poor	3	2
Bad	3	2
Total	21	21

SUMMARY AND CONCLUSIONS

Study of the transplantation of Tenon's capsule in postoperative cicatricial fixation of the eye was carried out experimentally in rabbits using different technics. The results were analyzed in 47 cases. The types of grafts used were: free, pedicle and apron. The substances were: Tenon's capsule, amnioplastin, egg membrane and peritoneum. Adhesions were produced by means of manipulation of the muscle, scarification of the sclera with a knife and abrasion of the muscle tissue with either a blunt or sharp instrument. The pedicle grafts produced better results probably because the graft was less firmly fixed in its final position and a smaller number of sutures were employed. From a mechanical standpoint the apron graft apparently forms a tunnel in which the muscle gains greater motility because conjunctival adhesions may be important.

After transplanting Tenon's capsule in rabbits' eyes there was never complete freedom from adhesions although motility was usually increased. This is possibly explained by the histologic structure of the capsule.

Amnioplastin, egg membrane and peritoneum proved unsatisfactory as substitutes for Tenon's capsule grafts.

The experiments showed that rough manipulation of the muscles or capsule alone produces adhesions. Therefore, it is of the utmost importance to remember in carrying out any muscle surgery that the greatest care be taken to prevent trauma to the muscle and Tenon's capsule. Abuse of the muscle with a muscle hook, knife, scissors or forceps can easily produce a fixation that requires secondary repair, which could probably be avoided if sufficient caution had been exercised by the surgeon. The traumatism and reaction from suturing and suture material should also be prevented if possible. In cases of complete fixation of the muscle to the sclera, motility is definitely improved by the transplantation of Tenon's capsule. It was felt that

two factors were important in effecting this improvement: (1) prevention of firm new adhesions and (2) the implanting of a pliable tissue for the muscle to work on.

Clinical reports of 4 typical cases are presented and a table showing the findings in the 21 operations is included. Definite improvement in convergence occurred in 16 of the 18 patients with postoperative cicatricial exotropia upon whom operations were performed. Limitation of motion improved in the majority of cases, being completely or partially corrected in 16 cases. Complete restoration of function was obtained in one case and marked improvement in 9. The postoperative deviation showed an average improvement of 50 per cent in the exotropic cases and of 66 per cent in the esotropic cases. Vertical imbalance was reduced to one-third of the preoperative state.

From our experience in the study of 21 operations we do not claim that the results are perfect, and in some cases the condition has been aggravated by the operation. Nevertheless, we believe that transplantation of Tenon's capsule for the correction of postoperative cicatricial strabismus is justified. The operation offers a good chance of improving the appearance and motility of the eyes of patients who have adhesions between their ocular muscles and the sclera and show marked mechanical limitation of motion in the field of action of the affected muscle.

REFERENCE

1. Berens, C.: Tenon's Capsule Transplants in Surgery of the Ocular Muscles, with Especial Reference to Postoperative Deviations with Adhesions Between the Muscles and the Eyeball, Am. J. Ophth. 21:536, 1938.

DISCUSSION

A. D. PRANGEN, M.D., Rochester, Minn.: This paper by Dr. Berens and Dr. Romaine is a valuable contribution to surgery of the extra-ocular muscles. First, it offers a new and feasible method for dealing with postoperative adhesions which interfere with motility. Second, and of most interest to me, it points to the significance of Tenon's capsule and the value of preserving its function when operations are performed on the extra-ocular muscles.

On considering the operation and its technic, I agree with the conclusions of the authors that the sliding flap or, better yet, the apron flap should be the best procedure. By this method, less tissue would be damaged, fewer sutures would be used, and the exposed muscle would be enclosed by its original loose fascial envelope through which it can slide. The results of this operation, as reported in this paper, are not extravagant, but are encouraging. When cases of limited postoperative motility from adhesions are encountered, this procedure should be tried.

In considering cases of limited postoperative motility from adhesions, one wonders why these complications occur. I believe they are caused either by unnecessarily damaging Tenon's capsule at the time of operation or by overdoing the original operation. The latter cause is obvious and, I assume, is not the cause of the cases of postoperative cicatricial loss of motility considered in this paper. The first cause, disturbed function of Tenon's capsule, is the point under discussion. Throughout the years I have watched muscle operations, my own and those of others, and I gradually have come to feel that coarse carpentry will not suffice on the delicate mechanism of the extra-ocular muscles. The regional anatomy and physiology of the several tissues involved in the function of the extra-ocular muscles must be understood and respected. Of these tissues, the fascia bulbi or Tenon's capsule is of paramount importance. My consciousness of the importance of Tenon's capsule was awakened by the late Dr. P. C. Jameson.

The studies on the anatomy and physiology of Tenon's capsule of rabbits' eyes made by Dr. Berens and his associates are most informative. They remind us that Tenon's capsule is a sheet of loose fibrous connective tissue enveloping the muscles, a synovial membrane through which the muscles slide. The authors state, "It is significant to note that by even slight manipulation of the muscle, as would be made in the usual resection or recession, numerous adhesions were produced without the aid of other implements." Postoperatively, every rabbit's eye showed adhesions of the muscle to the sclera, but these adhesions were less between Tenon's capsule and the sclera than between

other tissues and the sclera. Moreover, even when the capsule was adherent to the conjunctiva and the sclera, the loose structure of the capsule allowed the muscle to slip through it.

It seems clear, therefore, that in operating on the extra-ocular muscles surgeons should seek to effect minimal trauma to Tenon's capsule on the muscle proper and should try to keep the fascial envelope of the muscle intact during resection or recession. It is possible to do this with care. When the conjunctiva is opened Tenon's capsule on the muscle can be avoided. In isolating the tendon and muscle, sharp cutting dissection parallel to the muscle can be used instead of tearing blunt dissection. This latter tends to pull the capsule off the tendon and muscle, particularly if the capsule has been opened by the incision. The tongue of tendon and muscle with the intact capsule then can be altered en masse. This, as Dr. Berens and his associates have shown, would invite fewer scleral adhesions, and even though adhesions occur, the muscle will slide through its original capsule. I know this to be true from my own experience. It is the reason I must reject any operation which advocates destruction of Tenon's capsule when seeking to improve or preserve motility. As a further means of lessening the trauma of tissue and adhesions, I have come to use as few tendon sutures as possible and also fine No. 000 and No. 0000 plain gut sutures which cause less reaction but hold long enough for union to occur. Also, I avoid burying these sutures by tying the knots outside of the conjunctiva.

For cicatricial loss of motility I believe the operation, as advocated by Dr. Berens and his associates, is feasible and, as I said previously, should be tried. No doubt, for the reasons already stated, Tenon's capsule is the tissue of choice for correcting these adhesions when a transplant is needed.

CONRAD BERENS, M.D., New York: First, permit me to thank Doctor Prangen for his excellent discussion and for stressing the importance of Tenon's capsule in the ordinary, everyday muscle operations that we have to perform. His method of operating is one which I have tried to follow for some time, but I confess that since we have had postoperative cicatricial strabismus in one patient of mine, I have been doubly careful.

Doctor Prangen asked whether we were describing the ordinary over-correction. I have had and have reported many over-corrections, but not, I am glad to say, this type of deviation in which we found these extensive adhesions between the muscle and the eyeball with almost complete limitation of movement in the field of action of the affected muscle. One case is a low incidence of cicatricial strabismus in view of the fact that in the early days I was taught to cut into the capsule and to snip along the sclera and I did it rather freely. In the light of the experiments on animals that we have performed, I do not see why I have not had a great many more of these unfortunate complications.

DIFFERENTIAL DIAGNOSIS OF PARESIS OF THE OBLIQUES AND THE SUPERIOR AND INFERIOR RECTI MUSCLES

WILLIAM THORNWALL DAVIS, M.D.
WASHINGTON, D. C.

THIS PAPER WAS ILLUSTRATED BY A FILM, DEPICTING THE VARIOUS TYPES OF PARALYSIS OF THE OCULAR MUSCLES. THESE PICTURES WERE MADE FOR SLOW MOTION EXHIBITION SO THAT ONE COULD SEE MORE CLEARLY THE PHENOMENA ASSOCIATED WITH MUSCLE PARESIS.

I MUST ask your indulgence, in that I have exceeded the title of my paper with this little movie. After we had photographed the cases showing the differential diagnosis of paralysis of the superior rectus and paralysis of the superior oblique, several other patients were observed with a similar condition, so that we were able to make a series of pictures showing vertical disorders of motility. We made motion pictures of patients showing paralysis of the inferior oblique muscle and dissociated hyperphoria and took the liberty of putting them in this exhibit because it isn't often that one can get such patients for a motion picture.

Bielschowsky states, "By far the most frequent type of paralysis of a single vertical motor anomaly is trochlear palsy." The rarest one is paralysis of the inferior oblique. The most striking symptom of palsy of the oblique muscles, both superior and inferior, is habitual functional torticollis. Tilting of the head does not occur with palsy of the vertical recti. Until Bielschowsky formulated the differential diagnostic signs between palsy of the superior and inferior recti and the obliques, paresis of the vertically acting recti and of the obliques was frequently confused.

The outstanding differential diagnostic sign is as follows: In palsy of the right superior oblique or of the left superior rectus, when the patient looks up and to the left there is a high degree of right hyperphoria, due to overaction of the right inferior oblique. In palsy of the right superior oblique, if the head is tipped to the right shoulder, there is little or no vertical deviation. If there be palsy of the right superior rectus there is no change in the vertical deviation on tipping the head either to the right or left shoulder. There are other diagnostic signs, but the above-mentioned indication is the most easily observed and the most important. It requires no paraphernalia.

When there is a palsy of the right inferior oblique, the same difficulty obtains in distinguishing it from palsy of the left inferior rectus. When the eyes are rotated to the left, the right eye turns strongly down and in. This is caused by over-action of the right superior oblique, the antagonist of the right inferior oblique.

The simplest and most dependable diagnostic sign is tipping the head. When the head is tipped to the right shoulder, the side upon which the palsy exists, there will be slight or no vertical deviation of the eyes. If the head is tipped to the left shoulder, the maximum vertical deviation occurs. This is directly opposite to that which occurs in palsy of the right superior oblique where the maximum vertical deviation occurs on tipping the head to the side of the lesion. In palsy of the inferior oblique the maximum deviation occurs on tipping the head to the side opposite the lesion. If the left inferior rectus is paralyzed there is no difference in the

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vertical deviation on tipping the head to either shoulder.

The above description refers to pareses of long standing. In such cases the pareses of the obliques may have recovered, but the spasm or contracture of the obliques, which are the antagonists of the paretic ones, continues. In old paresis of the right superior oblique, tipping the head to the right shoulder still elicits the characteristic maximum vertical deviation. The same thing can be observed in an old paresis of the right inferior oblique upon tipping the head to the left shoulder.

In paresis of recent origin of these muscles, lack of function of the involved muscle may be observed upon rotating the eyes into its particular field. There may be observed also the characteristic cyclophoria. Bielschowsky, however, points out that the most important sign is the maximum deviation observed upon tipping the head. He puts very strong emphasis upon that.

(Dr. Davis made the following comments during the showing of the motion picture.)

This is the characteristic position, tilting of the head.

These are slow motion pictures so as to give you time to see it well.

Now the head is tipped to the right shoulder. That is the sign and symptom that I wanted very much to illustrate, because it is by that sign that we can make a diagnosis in a very few moments. If you desire to follow out the other tests, that is proper and right in order to verify the diagnosis. You may see that the right eye is elevated, even without the cover test, when the head is tipped to the right.

There is a good deal of repetition there, but it is done purposely.

Now with the head tilted to the left, you will notice that there is also no vertical deviation, or very little. Remember that Bielschowsky said the maximum deviation occurs on tilting the head to the right shoulder in cases of paresis of the right superior oblique.

These cases of disturbance of the obliques have been very interesting, because the diagnosis had always been so exceedingly difficult until Bielschowsky made it very clear. When paresis of the obliques is complicated with other forms of hyperphoria it is difficult to make a diagnosis or perhaps impossible. For instance, in a case of an old paralysis of the right superior oblique with an over-action of the inferior oblique, complicated with an alternating hyperphoria or dissociated hyperphoria, diagnosis may be impossible; that is, for me. I have no doubt it would be possible with Bielschowsky.

Differential diagnosis: In order to be sure that the ocular deviation is a neurogenic disturbance, other tests must be made to show that the deviation is the same in all positions of the head and also with all positions of the eyes.

Dissociated hyperphoria is largely due to poor fusion. Restoration of fusion is necessary if the condition is to be corrected. It very often complicates high degrees of lateral deviations, esotropia and exotropia; it may be very confusing in those cases. In such cases the lateral deviation must be corrected first, and then by orthoptic training the fusion is stimulated to its proper amplitude and the dissociated hyperphoria will be cured.

DISCUSSION

SANFORD R. GIFFORD, M.D., Chicago, Ill.: Doctor Davis has so well described and demonstrated the principal points in differential diagnosis of these conditions that there is little left to say. There should be no need to insist before this group upon the value of exact diagnosis in paralysis of the vertical and oblique muscles. One still sees, however, a number of patients whose disorder has not been diagnosed, or if diagnosed, still without sufficient conviction to bring about appropriate remedial measures. This is a pity, since few cases are more satisfactory than those of partial paralysis of a vertical or oblique muscle in which appropriate surgery is carried out. The proof of the diagnosis is in the surgical result, and few patients are more grateful than those who have received appreciable improvement in their binocular field with relief of long-standing discomfort. I think most ophthalmologists are similar in an intractible tendency to forget rules concerning paralytic squint. Forgetting some of them

DIFFERENTIAL DIAGNOSIS OF PARESIS OF THE OBLIQUES AND THE SUPERIOR AND INFERIOR RECTI MUSCLES

WILLIAM THORNWALL DAVIS, M.D.
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THIS PAPER WAS ILLUSTRATED BY A FILM, DEPICTING THE VARIOUS TYPES OF PARALYSIS OF THE OCULAR MUSCLES. THESE PICTURES WERE MADE FOR SLOW MOTION EXHIBITION SO THAT ONE COULD SEE MORE CLEARLY THE PHENOMENA ASSOCIATED WITH MUSCLE PARESIS.

I MUST ask your indulgence, in that I have exceeded the title of my paper with this little movie. After we had photographed the cases showing the differential diagnosis of paralysis of the superior rectus and paralysis of the superior oblique, several other patients were observed with a similar condition, so that we were able to make a series of pictures showing vertical disorders of motility. We made motion pictures of patients showing paralysis of the inferior oblique muscle and dissociated hyperphoria and took the liberty of putting them in this exhibit because it isn't often that one can get such patients for a motion picture.

Bielschowsky states, "By far the most frequent type of paralysis of a single vertical motor anomaly is trochlear palsy." The rarest one is paralysis of the inferior oblique. The most striking symptom of palsy of the oblique muscles, both superior and inferior, is habitual functional torticollis. Tilting of the head does not occur with palsy of the vertical recti. Until Bielschowsky formulated the differential diagnostic signs between palsy of the superior and inferior recti and the obliques, paresis of the vertically acting recti and of the obliques was frequently confused.

The outstanding differential diagnostic sign is as follows: In palsy of the right superior oblique or of the left superior rectus, when the patient looks up and to the left there is a high degree of right hyperphoria, due to overaction of the right inferior oblique. In palsy of the right superior oblique, if the head is tipped to the right shoulder, there is little or no vertical deviation. If there be palsy of the right superior rectus there is no change in the vertical deviation on tipping the head either to the right or left shoulder. There are other diagnostic signs, but the above-mentioned indication is the most easily observed and the most important. It requires no paraphernalia.

When there is a palsy of the right inferior oblique, the same difficulty obtains in distinguishing it from palsy of the left inferior rectus. When the eyes are rotated to the left, the right eye turns strongly down and in. This is caused by over-action of the right superior oblique, the antagonist of the right inferior oblique.

The simplest and most dependable diagnostic sign is tipping the head. When the head is tipped to the right shoulder, the side upon which the palsy exists, there will be slight or no vertical deviation of the eyes. If the head is tipped to the left shoulder, the maximum vertical deviation occurs. This is directly opposite to that which occurs in palsy of the right superior oblique where the maximum vertical deviation occurs on tipping the head to the side of the lesion. In palsy of the inferior oblique the maximum deviation occurs on tipping the head to the side opposite the lesion. If the left inferior rectus is paralyzed there is no difference in the

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vertical deviation on tipping the head to either shoulder.

The above description refers to pareses of long standing. In such cases the pareses of the obliques may have recovered, but the spasm or contracture of the obliques, which are the antagonists of the paretic ones, continues. In old paresis of the right superior oblique, tipping the head to the right shoulder still elicits the characteristic maximum vertical deviation. The same thing can be observed in an old paresis of the right inferior oblique upon tipping the head to the left shoulder.

In paresis of recent origin of these muscles, lack of function of the involved muscle may be observed upon rotating the eyes into its particular field. There may be observed also the characteristic cyclophoria. Bielschowsky, however, points out that the most important sign is the maximum deviation observed upon tipping the head. He puts very strong emphasis upon that.

(Dr. Davis made the following comments during the showing of the motion picture.)

This is the characteristic position, tilting of the head.

These are slow motion pictures so as to give you time to see it well.

Now the head is tipped to the right shoulder. That is the sign and symptom that I wanted very much to illustrate, because it is by that sign that we can make a diagnosis in a very few moments. If you desire to follow out the other tests, that is proper and right in order to verify the diagnosis. You may see that the right eye is elevated, even without the cover test, when the head is tipped to the right.

There is a good deal of repetition there, but it is done purposely.

Now with the head tilted to the left, you will notice that there is also no vertical deviation, or very little. Remember that Bielschowsky said the maximum deviation occurs on tilting the head to the right shoulder in cases of paresis of the right superior oblique.

These cases of disturbance of the obliques have been very interesting, because the diagnosis had always been so exceedingly difficult until Bielschowsky made it very clear. When paresis of the obliques is complicated with other forms of hyperphoria it is difficult to make a diagnosis or perhaps impossible. For instance, in a case of an old paralysis of the right superior oblique with an over-action of the inferior oblique, complicated with an alternating hyperphoria or dissociated hyperphoria, diagnosis may be impossible; that is, for me. I have no doubt it would be possible with Bielschowsky.

Differential diagnosis: In order to be sure that the ocular deviation is a neurogenic disturbance, other tests must be made to show that the deviation is the same in all positions of the head and also with all positions of the eyes.

Dissociated hyperphoria is largely due to poor fusion. Restoration of fusion is necessary if the condition is to be corrected. It very often complicates high degrees of lateral deviations, esotropia and exotropia; it may be very confusing in those cases. In such cases the lateral deviation must be corrected first, and then by orthoptic training the fusion is stimulated to its proper amplitude and the dissociated hyperphoria will be cured.

DISCUSSION

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has been more profitable than having learned them. For it is true that the text book picture of diplopia, as shown in nine neat little squares, is rarely met with in practice. It is seen only in cases of fresh paralysis, and is supplanted rapidly by that resulting from contracture or spasm of the opposing muscle. Doctor Davis has done well to describe this stage of superior oblique paralysis, in which the diplopia is not limited to the down and in field, but exists all over the field, and the prominent feature is the upshoot of the paralyzed eye in adduction, due to over-action of the inferior oblique. I would go further and say that this is the usual picture seen, and is common in cases in which the superior oblique has so far recovered that it is difficult to be sure of any limitation of motion down and in.

(Slide) This is a case of paralysis of the left superior oblique muscle with a typical head-tilting to the right. The eyes are about level.

(Slide) This shows the patient looking up to the right, with over-action of the left inferior oblique.

More practical than remembering rules, at least in my experience, has been a somewhat childish method by which the position and functions of the various muscles are visualized while looking at the patient. Before examining the field of diplopia, the deviations in various parts of the field on cover test are observed, as advocated by Bielschowsky, Duane and White, including observation while the head is tipped. Thus, with the action of each muscle in mind, one arrives at the muscle which must be paralyzed or overactive to give the deviation observed. The only rules worth remembering are that the deviation on cover test of the non-paralyzed eye is always greater than that of the paralyzed eye, and that a marked increase in deviation on forced tipping of the head nearly always means paralysis of an oblique muscle. I would add one more and say that the deviation is always greater in adduction in the case of paralysis of an oblique and always greater in abduction in the case of paralysis of a vertical rectus muscle.

Bielschowsky's priceless advice is always remembered, that, in vertical deviations, one should consider only the primary action of the muscles, as their secondary action may be neutralized by preëxistnig phorias. I dislike to think how many diagnoses I have missed when following the text book picture which indicated that paralysis of an oblique should result in homonymous diplopia because of the abducting action of the obliques. Almost as often as not, in acquired cases, a preëxisting exophoria causes the diplopia to be crossed or crossed in one field and homonymous in another. In congenital cases of trochlear paralysis, a convergence is more likely to develop, which may be marked enough to stimulate concomitant convergent squint with overaction of an inferior oblique. This is especially apt to occur when some degree of suppression has developed, as may happen in congenital paralysis, so that diplopia is not noticed. In a child old enough to cooperate, however, diplopia can nearly always be elicited during tests, and in smaller children, head-tilting will always suggest a congenital trochlear paralysis. The case of a patient with left trochlear paralysis whom I recently treated (slide) is typical. At the age of 14 she suppressed the image of the left eye so much that she never complained of diplopia. She was more comfortable, however, when tipping the head to the right shoulder, and diplopia could be elicited easily when attention was directed to it. In this case the upshoot of the left eye on adduction was the prominent feature, but there was left hypertropia of 15 prisms in the primary position. (She happens to be looking down and in, so that she shows limitation of the motion of the superior oblique also, but she shows a marked upshoot on looking straight to the right.) This has been reduced to 3 prism diopters of the left hyperphoria by myectomy of the left inferior oblique.

I have cared for a few patients with congenital trochlear paralysis in whom the convergent deviation had become so marked as to require surgical correction in addition to what was necessary for the vertical deviation. In such cases myectomy of the inferior oblique is usually performed first and is followed later by whatever procedure on the horizontal recti is indicated by the degree of squint.

As Doctor Davis has pointed out, the most difficult problem of diagnosis is between partial paralysis of a superior oblique and that of the opposite superior rectus. This is especially true when there is alternating fixation or fixation with the paralyzed eye. The relative position of the double images is the same in both conditions. Thus, in left trochlear paralysis there is left hypertropia which increases on looking to the right with the upper pole of the left image tipped to the right. In paralysis of the right superior rectus muscle there is right hypotropia or left hypertropia, increasing on looking to the right and with the upper pole of the right image tipped to the left, though usually only slightly. If the patient fixes with the right or paretic eye, the image of the fixing eye is seen

as erect and that of the left eye is tipped with its upper pole to the right. Usually the head-tilting test, as described, will show marked increase in deviation on tipping the head to the left side in left trochlear paralysis while if there is paralysis of the right superior rectus muscle the deviation will be unchanged or improved.

(Slide) This is another case of paralysis of the left superior oblique muscle with the head tilted to the right in the normal position and no deviation.

(Slide) An attempt is being made here to force the patient's head to the left, and he shows an upshoot of the left eye on looking to the right and with the head tilted.

When there is doubt, it is often of value to note the degree of deviation of each eye on cover test. In a recent case of paralysis of the right superior rectus muscle (slide), when fixing with the left eye deviation was very slight, while when fixing with the right eye, as shown in the slide, the left eye showed a marked upshoot under cover. This, of course, established the right eye as the paralyzed eye. Fortunately, myectomy of the left inferior oblique, which is indicated in right superior rectus paralysis, is of almost equal value in left trochlear paralysis with overaction of the left inferior oblique, so we may possibly be wrong in the diagnosis and still do the patient some good.

In examining for diplopia, which is seldom done until a tentative diagnosis by the cover test has been made, the value of the horizontal line should be emphasized. The images of the vertical line, usually employed, often overlap in such a way as to be fused by the patient. Images of the horizontal line, when tipped, are fused much less easily and hence the patient will give much more reliable answers as to the tipping of images. Its only disadvantage is the necessity for mentally rotating the tipped image 90 degrees in order to visualize the rotation of the eye.

(Slide) This is a picture of Bielschowsky's, illustrating paralysis of the left superior rectus muscle, and showing the picture in the corresponding fields. This shows the patient fixing with the paretic eye, and shows the marked upshoot of the non-paretic eye when fixing with the paretic eye. This shows him fixing with the non-paralyzed eye and very little deviation. This shows his normal movement of the right eye on looking up and right, and this shows the marked limitation of motion of the right eye on looking up and left.

(Slide) This is our horizontal line shown as it is used, and this shows the mental rotation of it, which one must make in order to visualize the paralyzed muscle, which accounts for the tipping. We remember, of course, that the tipping is always in the direction in which the paralyzed muscle should move the eye, so that when the left image is tipped with the upper pole to the right, as in this case, there must be a paralysis of a right rotator, or in this case, the left superior oblique.

(Slide) Once this trick of mentally rotating the image is learned, its advantage is obvious. Many persons are seen who will give no information as to tipping with the vertical line, but who do so readily with the horizontal line. The red-glass is seldom necessary in such tests and only serves to conceal the deviations which are occurring and which we wish to observe.

I have just a few slides showing this test.

(Slide) This is a case of paralysis of the left superior oblique muscle before operation, showing a very crude picture of the diplopia field (slide) and the next picture shows the binocular field after myectomy of the left inferior oblique.

(Slide) The next slide shows paralysis of the superior rectus muscle on the left side. When the patient is looking up and to the right one does not see any deviation. When she is looking up and to the left, there is a marked limitation of movement of the right eye on looking to the left in the field of the paralyzed left superior rectus muscle.

(Slide) The next slide shows paralysis of the inferior oblique muscle. This is from Bielschowsky, and it shows that the motion of the left eye is normal when the patient looks up and to the left, but on looking up and to the right there is complete paralysis of the left inferior oblique.

(Slide) The next slide is a case of mine in which one picture shows paralysis of the left inferior oblique.

MALIGNANT TUMORS OF THE MIDDLE EAR AND THE MASTOID PROCESS

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and
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INVOLVEMENT of the middle ear and the mastoid by a malignant neoplastic process has generally been considered almost a pathologic curiosity. Moreover, the condition has been almost universally supposed to respond unfavorably to therapeutic measures. Actually, such lesions occur less rarely than the medical literature would lead one to believe and, if the condition is recognized reasonably early and is dealt with adequately, the prognosis is as satisfactory as that generally offered by malignant tumors of the nasal accessory sinuses.

LITERATURE

The literature on malignant tumors of the middle ear and the mastoid process is not extensive. The largest series of cases reported (thirteen) is that of Thorell,[9] presented with a consideration of the treatment of this condition at the Radiumhemmet. Outstanding articles on this subject in the American literature have been written by Furstenberg,[2] Schall[5] and Spencer.[7] In addition, reports of cases have been presented by Robinson,[4] Bowman,[1] Smith,[6] Newhart,[3] Stokes,[8] and others.

INCIDENCE

During the last twenty years (January 1, 1922 to December 31, 1941) there were seen at the Mayo Clinic forty-eight patients who had malignant tumors involving the middle ear and the mastoid process. More than 500 additional patients observed during this period had malignant lesions involving only the pinna or the external auditory canal. The incidence of malignant tumors of the middle ear and the mastoid among registrants in the clinic during the last six years has been approximately 0.003 per cent.

This study is based on a group of thirty-eight cases in which treatment was carried out in the clinic; excluded were ten additional cases in which the process of disease was so extensive that therapeutic measures would obviously have been of little avail. Excluded also was one case the clinical and operative findings of which, together with the subsequent course, failed to substantiate a histopathologic diagnosis of epithelioma made on tissue removed from the external auditory canal and from the mastoid antrum at the time of operation. The patients were divided equally as to sex, there being nineteen males and nineteen females. The ages of the males ranged from twenty-seven to seventy-one years and of the females from three to eighty years. Nineteen patients (50 per cent) were between fifty and seventy years of age, thirty (79 per cent) were between forty and seventy years. Five patients were less than forty years old and three were seventy years or older. The left ear was involved in twenty-two instances, the right in sixteen (fig. 1*a* and *b*).

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FIG. 1 — A man, aged fifty-four years. *a*. Recurring basal cell and squamous cell epithelioma, grade 1, measuring 3.5 by 8 cm.; the lower portion of the lesion was ulcerated deeply, involved the auricular cartilage and clinically appeared to be invading the mastoid. Removal was effected with electrocoagulation and radium applied interstitially. It was necessary to sacrifice the lobe of the ear and a large portion of the concha. Neither the middle ear nor the mastoid was involved. The patient was not included in the series on which this report is based. *b*. Result six and a half months after treatment. At the time of writing, patient has been well more than three years.

SITE OF ORIGIN

The middle ear and the mastoid may be involved by a malignant neoplasm arising primarily in these situations or they may be invaded secondarily by a lesion that has originated in the pinna or in the structures adjacent to it. For convenience we shall designate the former as intrinsic growths and, because it frequently is impossible to determine whether the pathologic process has developed in the middle ear or in the deeper portion of the external auditory canal, growths springing from this latter region are included in this category. Tumors having their origin in the pinna, in the skin of the surrounding region, in the parotid gland, in the nasopharynx or in other structures outside of the ear are referred to as extrinsic. In our experience the intrinsic growths outnumber the extrinsic and this predominance appears to hold generally in the cases of this type reported by other observers. In twenty-five of the thirty-eight cases in this series the tumors were considered intrinsic. In the remaining thirteen cases the lesions were extrinsic.

SYMPTOMATOLOGY

The clinical course of malignant disease of the middle ear and the mastoid varies greatly and the symptomatology is accordingly not the same in all cases. Among factors influencing the course of the disease and, thereby, the symptoms complained of are the site of origin of the neoplasm, the activity and extent of the growth, the presence of inflammation, trauma, treatment and the age and general condition of the patient. In the presence of inactive lesions about the pinna or canal that have not become infected or have not been subjected to trauma, symptoms frequently are mild and such growths may be present for years without causing the patient sufficient trouble to bring him to a physician.

On the other hand, highly malignant neoplasms, either intrinsic or extrinsic, are likely to produce severe symptoms promptly, especially if subjected to trauma or to incomplete treatment. Symptoms may be present for only a few weeks or months although they may exist for some years. In general, symptoms were present for a much shorter period in the group of cases of intrinsic neoplasms than in the group of extrinsic neoplasms. As might be expected, some of the inactive extrinsic growths produced the mildest symptoms and were of longest duration.

Chronic otorrhea, often of many years' duration, may precede the development of a malignant neoplasm in the middle ear. The presence of this discharge frequently renders it difficult if not impossible to determine even the approximate date of onset of symptoms caused by the malignant lesion. In a few of the cases of intrinsic neoplasms, polyps had been removed from the external auditory canal on repeated occasions during the course of several years preceding examination at the clinic but in most instances microscopic studies of the excised tissue had not been carried out and the approximate time of inception of the malignant process could not be determined. In only four of the twenty-five instances of intrinsic malignant neoplasm did it appear likely that cancer had existed for as long as five years. One patient, a woman aged forty-seven years, had had a chronic discharge from the ear since the age of ten years. Eight years prior to her coming to the clinic a polyp had been removed from the external auditory canal, a diagnosis of "tumor" had been made after microscopic examination and radium had been applied. Tissue removed at the clinic proved microscopically to be squamous cell epithelioma, grade 4. It is impossible to conceive of such a highly malignant condition having been present for more than a few months. In eleven of the thirteen cases of extrinsic growth, carcinoma had apparently been present for more than five years and in seven of these for ten years or more. The longest period of duration was seventeen years; the patient was a man fifty years of age who had an adenocarcinoma of mixed tumor type originating in the parotid gland and only in recent years secondarily involving the mastoid. All of the remaining lesions of long duration in this group of extrinsic neoplasms were adenocarcinomas or basal cell epitheliomas.

The symptoms most commonly complained of by patients who have malignant tumors of the middle ear and the mastoid are pain, discharge from the ear, the presence of an ulcer in or about the ear and decreased hearing on the affected side. Bleeding from the ear, vertigo, facial paralysis and the presence of a tumor are encountered less frequently. Mastoid tenderness, tinnitus, ankylosis of the mandible and difficulty in swallowing and speaking due to paralysis of the soft palate, pharyngeal constrictors and tongue may be causes of complaint. The complaint most commonly made by patients in our series was of pain in the ear. A few patients, however, experienced no pain whatever. In a number of instances, pain was present also in the mastoid, temporal, occipital and frontal regions or it was referred entirely to these parts. It was usually of a steady aching character but occasionally became lancinating and in some cases was more of a sense of pressure or throbbing than intense distress. At times the pain was severe, and at intervals of a few weeks acute pain occurred, lasting for periods of a week or ten days. A few patients noted that their pain was constant but was worse at night and was aggravated by lying down, by moving the ear and by cessation of the aural discharge. In some instances it came on at about the same time each day.

TABLE 1

Symptoms complained of most frequently by 38 patients

Symptom	*Patients*
Pain	33
Discharge	22
Ulceration	15
Deafness	12
Bleeding	8
Facial paralysis	7
Vertigo	6
Mastoid tenderness	3
Tinnitus	3

Next to pain in or about the ear, complaint was made most commonly of discharge from the ear. This was present in twenty-two of the thirty-eight cases. At times it had had its inception in an acute otitis media in childhood and thus had preceded other symptoms by years. Often there was nothing unusual about the character or the amount of the discharge but at times its very foul odor, watery consistency and the fact that it was blood-tinged had been noted by the patient. A fifth of the patients had experienced spontaneous bleeding from the canal. The number of instances in which the more common symptoms were encountered is shown in table 1.

Among the thirteen patients who had extrinsic lesions the presence of an ulcer or a tumor in the external canal or about the pinna or the mastoid region was the most frequent cause of complaint. In a number of cases radical treatment had been carried out previously so that the typical appearance of the neoplasm had been lost and a clinical diagnosis could not be made immediately. Extensive ulceration was present in some of the cases and in an occasional case an ulcerated lesion was fixed to the mastoid process, perforated the pinna or was destroying a varying portion of the auricle. In one instance the entire pinna had been destroyed by the ulcerative process.

The duration of the aural symptoms prior to the patient's coming to the clinic varied a great deal depending on the nature of the lesion, its activity and the site of origin. It was longest in the cases of adenocarcinoma of mixed tumor type and especially in those in which the tumor arose outside the ear. It was shortest in cases of the more active squamous cell epitheliomas, particularly in those of the intrinsic group. Next to the cases of adenocarcinoma in duration of symptoms were the cases of basal cell epitheliomas.

ETIOLOGIC FACTORS

In a large percentage of the reported cases of malignant tumors of the middle ear and the mastoid the lesions were preceded by otorrhea of long duration. This is a condition which is considered generally an important etiologic factor in the development of such neoplasms, although whether it has any direct bearing in this regard is debatable. It seems logical to assume, however, that the constant presence of an aural discharge might well cause cancerous change in the tissues thus irritated, just as habitual moistening of the lips by the tongue has appeared at times to initiate labial epithelioma. It is possible only to theorize in this connection for other factors must play an important role as is evidenced by the fact that tumors of this type frequently develop entirely independently of any such irritation.

In the series of thirty-eight cases studied there had been long-continued aural discharge in only eleven. It is of interest that in six of these eleven cases the site of origin of the malignant process was apparently in the external canal, whereas in at least two of the other five the neoplasm originated within the tympanum. The remaining three growths were so extensive that the point of their inception could not be ascertained. A number of cases of extrinsic lesions were included in this series although the presence of a chronic discharge in association with such lesions probably is of little significance. Six of the eleven

neoplasms developing in the presence of a persistent otorrhea were squamous cell epitheliomas, three were hemangioendotheliomas, one was an adenocarcinoma and one was a fibrosarcoma. Other conditions encountered having a possible causative bearing were ichthyosis of the canal occurring in one case and a chronic pruritus, present for twenty-five years, of the external canal in another. Two patients had experienced repeated attacks of erysipelas, and in one of these cases, in an ulceration persisting after the acute illness, malignant change had developed subsequently.

CLINICAL FINDINGS

TABLE 2

Clinical findings in 38 cases of malignant tumors of the middle ear and the mastoid

	Cases
Involvement of external auditory canal	28
Discharge	22
Tumor in canal	15
Obvious malignancy	15
Ulceration	15
Fixation of lesion over mastoid or to canal wall	10
Facial paralysis	7
Perforation of pinna	4
Mastoid tenderness	3

The clinical picture presented by cases of malignant tumors of the middle ear and the mastoid varies greatly depending on whether the neoplasm has originated within these structures or whether it has originated externally and is invading them secondarily (table 2). Tumors developing intrinsically may reveal little evidence of their presence until well advanced, in most instances the subjective symptoms being out of all proportion to the physical findings. In an unusual case of this type, the patient a woman forty-three years of age, the only objective sign demonstrable was the presence of tenderness in the tissues over the mastoid region, yet exploration revealed an extensive highly malignant squamous cell epithelioma of the mastoid with considerable destruction of bone. More commonly the external canal is filled with polyps which frequently are recurring and may be fairly fibrous. A highly vascular, readily bleeding mass or a granular growth may be present in the canal or the canal may be markedly narrowed by diffuse thickening or infiltration of its walls. A profuse, pulsating, foul discharge which is at times watery and blood-tinged may be associated with these findings (figs. 2*a* and *b*).

In cases of extrinsic malignant lesions with invasion of the tympanum and the mastoid, the clinical findings are likely to be pronounced unless treatment has been carried out recently; in a high percentage of cases the nature of the condition will be obvious to a physician who has had appreciable experience with malignant neoplasms. A clinical diagnosis of epithelioma was made in a large proportion of the cases of extrinsic lesions in this series and subsequent microscopic studies corroborated this determination. In a few cases the neoplasm was recognizable on inspection as carcinoma of mixed tumor type. Malignant ulceration of varying extent was present in most of the cases of epithelioma and in a number there was fixation of the ulcer over the mastoid region. Direct extension of the neoplasm inward along the external auditory canal was encountered usually although at times the ulceration overlying the mastoid was penetrating into the bony structures beneath. Perforation of the pinna had occurred in a number of cases and varying portions of the auricle had been destroyed by the tumor. In one case there was complete loss of the pinna (fig. 3*a*, *b* and *c*).

PREVIOUS TREATMENT

All but two of the patients in this series had received treatment for aural disease prior to coming to the clinic. This treatment had ranged from use of

FIG. 2a and b. — Adenocarcinoma, mixed tumor type, of ear and mastoid. Note fullness in the parotid and mastoid regions. The tumor was removed through a postauricular incision; although its outer portion was encapsulated, it was infiltrating in its deeper portion; excision was supplemented with implanted radium; no recurrence eight and a half years later.

FIG. 3 — A woman, aged fifty-nine years. *a*, Recurring basal cell and squamous cell epithelioma, grade 1, of left ear and mastoid; *b*, note perforation of pinna by the lesion. Treatment consisted of electrocoagulation, radical mastoidectomy and irradiation. *c*, Result two and a half years later. The patient had temporary postoperative facial paralysis. At the time of this report she has been well three and three-fourths years.

ear drops and local application of antiseptic solutions, ointments and caustic to removal of polyps, excision of tumors, irradiation and mastoidectomy. Roentgen therapy had been used in twelve cases and radium in thirteen. Most of these patients had had extrinsic lesions. Polyps had been removed from the external auditory canal in seven cases but in few of these had the tissue been subjected to microscopic examination. Mastoidectomy had been performed in six cases and in three of these the malignant nature of the condition had been recognized. Three of the patients had undergone mastoidectomy at the clinic from two to sixteen years prior to our establishing a diagnosis of malignant disease. One of these patients had had two operations on the mastoid, ten and sixteen years respectively prior to recognition of the malignant condition. At the time this paper is written this patient and another have been well for periods of eight and ten years respectively, while the third has not had recurrence after two years (fig. 4*a* and *b*).

FIG. 4 — *a*. Right-sided facial paralysis produced by fibrosarcoma, grade 4, of middle ear, external auditory canal and nasopharynx. Appearance of patient almost seven years after eradication of the neoplasm by means of mastoidectomy, radium and roentgen therapy. *b*. Result following correction of facial paralysis by insertion of fascia lata loops and plastic.

ROENTGENOGRAPHIC STUDIES

Roentgenograms of the mastoid should be made in all cases of chronic infection of the ear and in those in which there is a question regarding the possibility of a neoplasm in this situation. This is particularly true in cases of intrinsic lesions since in these the roentgenograms frequently will furnish considerable information that cannot be obtained from physical examination alone. Roentgenographic studies are also essential in cases of extrinsic tumors that extend into the external auditory canal or are fixed to the mastoid process. Although roentgenograms at times fail to reveal early involvement of bone in this group of neoplasms they often will help in determining the extent of the process

if invasion of bone is present. Since in tumors of the intrinsic group the process usually originates within the tympanic cavity or in the mastoid, roentgenographic evidence of destruction of bone would be expected in a higher percentage of cases than in the extrinsic group. As a matter of fact, the contrary was noted in our series. Among the twenty-five cases in which the malignant process arose within the mastoid cavity, middle ear or deep in the external auditory canal, the roentgenograms revealed evidence of pathologic change in the mastoid in twenty-one (84 per cent). Destruction of bone was evident in eight cases (32 per cent), cloudiness in seven (28 per cent), sclerosis in seven (28 per cent) and a postoperative defect in four (16 per cent). Among the cases in which the primary involvement was in the pinna or the tissues adjacent to it and in which the middle ear and mastoid were invaded secondarily roentgenograms were positive in nine (69 per cent). Destruction of bone was shown in six cases (46 per cent) and cloudiness in five (38 per cent). In four cases (31 per cent) some cloudiness of the mastoid was present but destruction of bone could not be discerned.

TREATMENT

In the past the treatment of malignant tumors of the middle ear and the mastoid has been discouraging. Such cases are encountered so infrequently that it has been difficult to develop an effective method of therapy based on accumulated experience. Treatment has consisted chiefly of surgical removal and irradiation either alone or combined and in only the exceptional case has this resulted in more than temporary palliation. Several factors have contributed to this unfavorable response to treatment. Among these are (1) the delay in establishing a diagnosis, (2) the difficulty of access to tumors involving the inner portion of the external auditory canal, (3) the frequent impossibility of determining clinically the extent of involvement, (4) the timidity of the surgeon with regard to carrying out radical measures, and (5) the ineffectiveness of radium and roentgen therapy in controlling malignant neoplasms that are invading either osseous or cartilaginous structures. These various factors were concerned in the unfavorable results observed in a number of the earlier cases in this series and attention to them has greatly improved the outlook.

Originality is not claimed for the method of treatment that we have employed in cases of this type. The procedure has evolved pretty largely from that used in dealing with malignant neoplasms of the nasal accessory sinuses and larynx and, like the latter, has improved considerably since the development of electrosurgical methods. In the earlier cases treatment was given almost exclusively with radium packs and direct application of radium tubes or a plaque with or without excision. This method of treatment has been supplanted by the more rational and effective procedure of exposing the tumor surgically, removing it with electrocoagulation under direct observation and supplementing this with implantation of radium points directly into the wound and application of radium packs or roentgen therapy externally. The results obtained with this form of treatment well bespeak its effectiveness. An exceptionally lucid exposition of the problems involved and the indications for treatment in these cases was presented by Furstenberg,[2] in 1924, in reporting a case of primary carcinoma of the middle ear and the mastoid. We quote from his paper: "In the department of otolaryngology of the University of Michigan the following principles are rigidly adhered to in the surgical treatment of malignancy. First, radical excision of the neoplasm and removal of all that kind of tissue where it has originated

or invaded and second, the employment of a technic that will leave the site of the new growth open to inspection for the remainder of the patient's life. The radical mastoid operation lends itself to these arbitrary requirements in that it permits of radical excison of the growth and leaves the middle ear and mastoid cavity accessible to future cauterization in the event of recurrence."

Surgical procedure. — The wide removal of the accessible portion of the neoplasm by means of electrocoagulation, either the coagulating current or the cutting current or both being employed, seems definitely to be an improvement over excision. Often the tumor is fixed to the mastoid process and must be stripped directly from the bone. If there is any question regarding the possibility of the bone being invaded, the involved portion should be removed and the mastoid opened. Perforation of the pinna may be present, in which event it is necessary to sacrifice enough of the full thickness of this structure to include a wide margin of the normal tissue. When, as is commonly true, the growth fills the external auditory canal or extends deeply along its walls electrocoagulation is carried as far as it is possible to visualize the tumor. The mastoid then is opened and, unless neoplasm is encountered, the procedure at once is carried through to completion as a radical mastoidectomy, the posterior canal wall being taken down. If tumorous tissue is encountered on removal of the bony cortex, which is often the case, this tissue is destroyed with the diathermy electrode, the dissection of bone being continued as necessary to expose more of the neoplasm. In this way electrocoagulation and dissection of bone are alternated until the tumor has been completely removed. During this process a varying portion of the dura may be exposed. In fact, a wide area of dura and a considerable part of the lateral sinus, the jugular bulb and other intracranial structures may be uncovered. At times an extension of the tumor is found directly in contact with the dura or sinus and there may be malignant infiltration of these parts.

When this is true the possibility of effecting complete removal of the pathology is practically nil. Fortunately, much more often, even though the tumor is highly malignant, a thin capsule will be found separating it from the dura and, provided the remainder of the growth can be cleanly removed, the primary lesion can be entirely eradicated. As the bony posterior wall of the external auditory canal is removed, if this wall has not already been destroyed by the pathologic process, the portion of the tumor that could not be reached through the meatus is rendered accessible.

At times a tumor that is well encapsulated on its outer surface will be found infiltrating in its deeper portion. The contrary may also be true. Both of these contrasting conditions have been seen in cases of adenocarcinomas of mixed tumor type in this situation. Provided the dura is intact, surprisingly little postoperative reaction usually follows the surgical procedure even though the neoplasm has been extensive and has necessitated intensive electrocoagulation. There is no doubt that in questionable cases the risk of meningitis and of other intracranial complications is increased greatly by attempts to deal with such conditions more conservatively and by failure to open the mastoid and to expose the dura. After surgical intervention the patient as a rule experiences marked relief from pain and, if the operation has been complete, cessation of the otorrhea is phenomenal. However, if the neoplasm is infiltrating the dura, any relief from symptoms produced by removal of the bulk of the tumor is likely to be only transitory, and prompt recurrence is almost sure to occur even

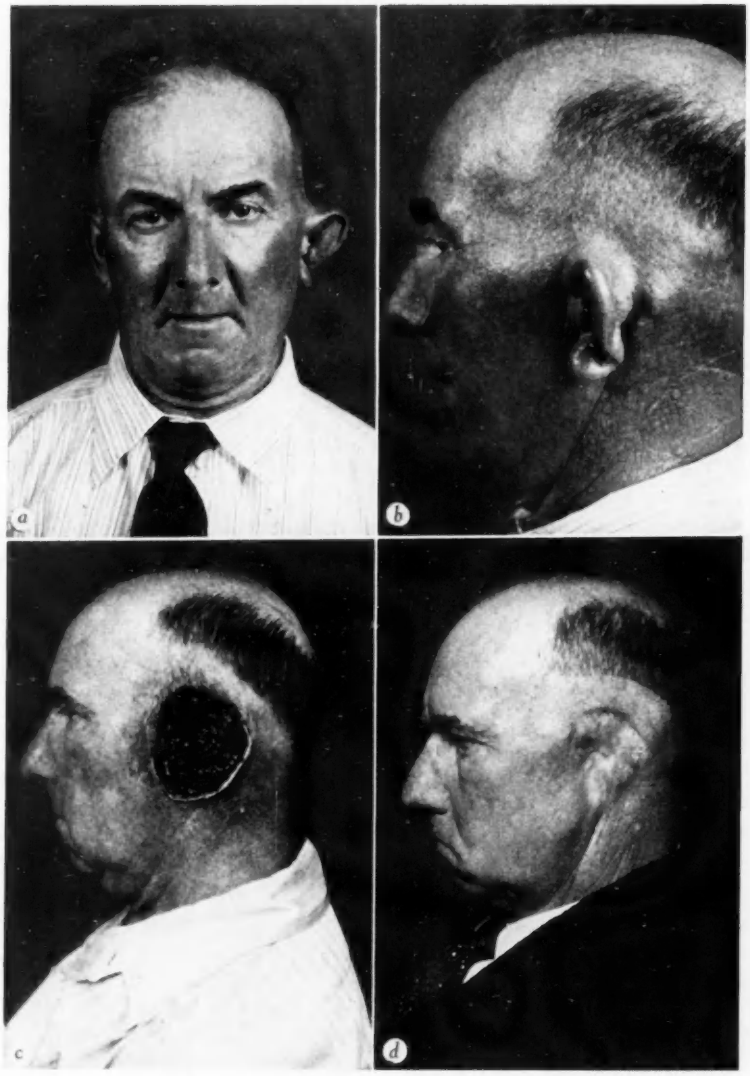

FIG. 5*a* and *b*—Recurring basal cell and squamous cell epithelioma, grade 1, of left ear and mastoid region. Note neoplastic infiltration of pinna and tumor protruding from meatus. *c*. Extensive defect resulting from treatment, consisting of electrocoagulation, radical mastoidectomy and irradiation, required to remove the tumor. Sacrifice of the facial nerve and of the entire auricle was necessary. *d*. Healing complete. Patient at the time of this report has been well one and a half years after treatment. No neoplasm was demonstrable in the middle ear and mastoid; accordingly this case is not included in the series but is presented to demonstrate the radical treatment at times required to control malignant tumors in this situation.

though intensive irradiation is used to supplement the operation (figs. 5*a*, *b*, *c* and *d*).

It has been our custom routinely after surgical removal of a malignant neoplasm of the middle ear and mastoid to place radium points or tubes directly in the operative cavity and to give a fairly caustic dose. If removal of the tumor appears to have been clean, points containing 1 mg. each of radium element encased in platinum usually are employed, from ten to fifteen of these being held in place by means of iodoform gauze which keeps them well removed from the bone itself. These points remain in the wound for from twenty to thirty hours, a total dosage of 300 to 400 mg. hours being administered. A few days after operation this treatment is supplemented with radium packs, an additional dose of from 4,000 to 18,000 mg. hours being given. When the mastoid is opened, if the tumor is found to be highly malignant and the involvement so extensive that complete surgical removal appears impracticable, no attempt at complete surgical exenteration is made. Instead, radium points or tubes are immediately implanted directly into the growth and the wound either is sutured loosely or a gauze pack is inserted to hold the radium in place. This latter procedure has at times been found necessary in cases also in which an extensive highly vascular hemangioendothelioma is encountered on exploration of the mastoid. In some of the cases in our series the bleeding was uncontrollable and it was necessary to terminate the operation. Accordingly, insertion of radium and packing or suturing the wound was carried out.

Considerable necrosis of bone usually results from electrocoagulation and irradiation. In the mastoid the resultant sequestrum separates slowly, requiring a much longer period of time than that necessary after similar treatment inside the mouth. Whereas sequestra about the jaws usually separate and can be lifted out readily within two months, those involving the mastoid process and the temporal bone frequently require a year or longer. Because it often is impossible to determine the extent of the necrotic process, it is inadvisable to attempt to remove sequestra before they have loosened, for excessive trauma is likely to result from too early removal and there is a possibility of producing severe acute inflammatory reaction with extension of the necrosis, and even meningitis. On the other hand, if removal is delayed until separation is complete the process will be found well walled off, there is no appreciable trauma and epithelization of the wound takes place promptly, even though a wide area of dura may be uncovered. Should evidence of recurrence of the neoplasm appear at any time after the operation, however, removal of the sequestrum and biopsy may become necessary even though the dead bone still is fixed firmly (fig. 6*a* and *b*).

PROGNOSIS

In cases of malignant tumors of the middle ear and the mastoid the malignant process extends by direct contiguity; metastasis rarely occurs and gland dissection is not indicated unless the glands become involved. Extension into the cervical lymph nodes took place in only two of the cases in the series under consideration. In one of these cases a basal and squamous cell epithelioma, grade 1, had been present for years and recurred repeatedly prior to development in the glands. In the other case an adenocarcinoma (cylindroma type), grade 2, has been removed at the clinic on two occasions. There was no local recurrence of the malignant process at the time the patient returned with several metastatic nodes in the neck two and a half years following the last operative procedure. Block dissection was carried out.

The result likely to be obtained in any given case of malignant tumor of the middle ear and the mastoid is difficult to anticipate because of the great variation in the factors involved. The nature of the neoplasm, its activity and extent are extremely important and frequently it is impossible to determine these until the mastoid and the tympanum are explored. Many times the tumor will be found so extensive and highly malignant that palliation only can be expected. On the other hand, a growth that has appeared clinically to be bordering on inoperability may be found on exploration to be readily removable. This has been noted especially in cases of adenocarcinoma of mixed tumor type. In our experience one of the most frequently encountered intrinsic growths is the hemangio-endothelioma (angio-sarcoma) which, although it usually is of a low grade of malignancy, is so highly vascular that clean surgical removal often is impossible. In fact, in many cases electrocoagulation will not control the bleeding and coagulation cannot progress because of it. Although the results in four of the seven cases of tumors of this type included in our study have been satisfactory to the time of writing of this report, it seems certain that a better chance of entirely clearing up the pathologic condition would have been afforded had it been possible to remove the tumors by means of electrosurgical procedure and then to supplement this operation with irradiation, rather than to depend entirely on implanted radium and radium packs or roentgen therapy, as was necessary.

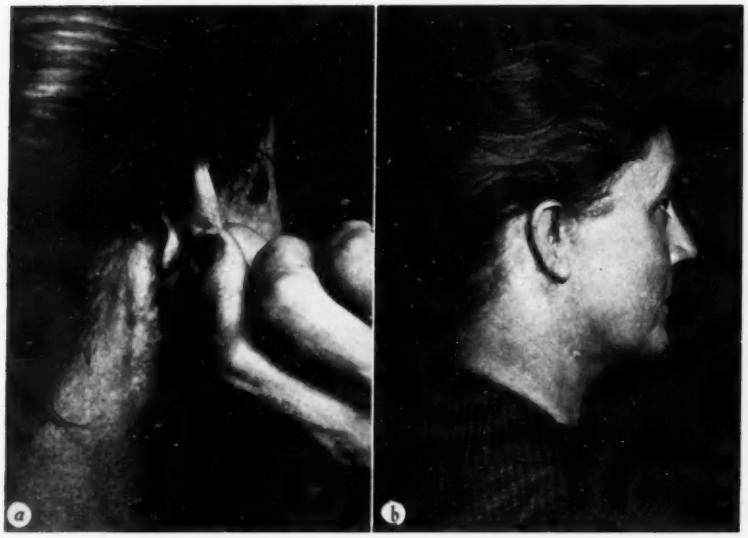

FIG. 6*a* — Scarring and defect following removal of recurring adenocarcinoma, grade 2, of the right ear and mastoid by means of electrocoagulation, radical mastoidectomy and irradiation two years previously. *b*. Patient returned approximately one year subsequently without local recurrence but with involvement of three right cervical lymph nodes for which a block dissection was done.

The combination of therapeutic measures now carried out in all cases of this type in which the extent of the tumor and the condition of the patient do not contraindicate, consisting of radical mastoidectomy, electrocoagulation and irradiation, is considered decidedly superior to the surgical excision and irradiation or irradiation alone previous-

ly employed. Good results were obtained with radium and the roentgen rays a few times, as in one case of fibrosarcoma, grade 4, of the external canal, middle ear, mastoid, nasopharynx and nasal fossa with an associated jugular foramen syndrome and unilateral facial paralysis. The patient was a woman thirty-eight years of age. After radium packs and roentgen therapy and radium on an applicator in the nasopharynx had been used, the neoplasm entirely disappeared and a plastic operation to correct the deformity incident to the facial paralysis was carried out some six years later. In general, however, palliation only is to be anticipated in such cases with this form of treatment. Even surgical removal of the lesion supplemented with irradiation but without radical mastoidectomy does not offer the patient the chance of cure that is his with the therapy now employed. Although some of the patients in our series who had malignant tumors in the middle ear and the mastoid lived for more than eight, and one for even fifteen years after they first were treated at the clinic, they had repeated recurrences and eventually succumbed to the malignant process. The results in several such instances are not considered satisfactory, for in retrospect and in the light of experience with more recent similar cases it seems likely that had radical mastoidectomy, electrocoagulation and irradiation been carried out, some of these patients would have had an excellent chance of complete cure. Moreover, the two patients in the series who died of meningitis and septicemia after surgical operation might well have been saved had the mastoid and the tympanum been exenterated, the dura uncovered and chemotherapy employed (fig. 7).

FIG. 7 — Patient well eight and a half years after treatment of hemangio-endothelioma, grade 2, of right middle ear and mastoid. Residual facial paralysis of right side developed spontaneously.

RESULTS

A summary of the results obtained in the thirty-eight cases in this series in which treatment was given is presented in table 3. It will be noted that twenty patients lived two years or more after treatment, fifteen lived three years or more, ten lived seven years or more, eight lived eight years or more, three lived more than ten years and one lived fifteen years.

We have been unable to obtain information from three of the patients in the series since they returned home after their original treatment at the clinic. It seems likely that one of these patients, a Negro forty-two years of age, may be well as his lesion, a squamous cell epithelioma, grade 1, appeared to have been cleanly removed. The other patient, a woman eighty years of age, had an extensive squamous cell epithelioma, grade 2, and in all probability the radium packs employed produced only tempo-

rary palliation. It has been impossible to trace a third patient, since her sequestrum was removed at home four months after dismissal from the clinic. In six cases the malignant process was extensive and palliation only was expected. All of the six patients succumbed to the disease within a few months to four years but it is interesting that one of these patients was carried along for four years by means of radium packs even though at the time of mastoidectomy the squamous cell epithelioma, grade 1, which filled the mastoid cells also involved the dura. At the time this paper is written two patients probably have recurrences, one after eight months, the other after fourteen months. One patient was living and free from the adenocarcinoma of his middle ear eight and a half years after removal of the lesion. Death, which occurred more than ten years after operation, probably was not due to the carcinoma. Another patient who had a recurring adenocarcinoma that appeared to be bordering on inoperability remained free of all symptoms and carried on his work as a locomotive engineer for eight years. He then returned with an extensive recurrence of the neoplasm which again was removed, but it recurred and produced a fatal termination three years later. Fourteen patients were living and well when last heard from, from sixteen months to ten and a half years after treatment. In three cases operation was carried out less than six months prior to this report and accordingly the outcome is indeterminate.

SUMMARY AND CONCLUSIONS

1. Thirty-eight cases of malignant tumors involving the middle ear and the mastoid are reported.

2. Such lesions occur more frequently than the medical literature would lead one to believe and the prognosis is more satisfactory than generally is thought.

3. Primary malignant (intrinsic) involvement of the middle ear and mastoid by a neoplasm arising in these situations occurs more frequently than does secondary involvement by a tumor originating in the pinna or in structures extrinsic to the ear.

4. The symptoms and clinical features of intrinsic malignant disease at times are the same as in cases of inflammatory disease of the middle ear and the mastoid and exploration and biopsy may be necessary for diagnosis.

5. The most effective forms of treatment for neoplasms of this type is a combination of electrocoagulation, radical mastoidectomy and irradiation and in indicated cases this treatment often is of considerable benefit.

6. At the time of writing, a number of the thirty-eight patients in this series have been living and well for periods ranging from three to ten years after treatment.

TABLE 3 *Summary of Results in 38 Cases of Malignant Tumors of the Middle Ear and the Mastoid*

Date of registration	*Age, years, and sex*	*Site of origin of tumor*	*Pathologic Condition*	*Treatment*	*Freedom from recurrence, years*	*Result*
Oct., 1919	54 M	Extrinsic	Basal cell and squamous cell epithelioma, grade 1	Repeated cautery excision and irradiation	Periods, 3½ to 5½	Died of recurrence 15 years after original treatment at clinic
April, 1921	28 F	Intrinsic	Fibrosarcoma, grade 4	Mastoidectomy and irradiation. Plastic correction fascial paralysis	8	Living and well more than 8 years
Sept., 1922	71 M	Extrinsic	Basal cell epithelioma	Irradiation		Palliation. Died of epithelioma May, 1924
Jan., 1923	57 M	Extrinsic	Squamous cell epithelioma, grade 3	Irradiation		Palliation. Died of recurrence May, 1927
March, 1924	51 M	Intrinsic	Squamous cell epithelioma, grade 2	Irradiation	3	Receiving colloid gold at home 3½ years later
Oct., 1924	43 F	Intrinsic	Squamous cell epithelioma	Exploration, mastoidectomy and irradiation		No benefit. Died of abscess of temporal lobe 3 months later
Jan., 1925	55 M	Extrinsic	Basal cell epithelioma	Radical mastoidectomy-removal	1	Probable recurrence 14 months later
March, 1926	51 F	Intrinsic	Squamous cell epithelioma, grade 2	Exploration and irradiation		Probably palliation only. Died; date unknown
April, 1928	50 M	Extrinsic	Adenocarcinoma, mixed tumor	Excision May, 1928 and April, 1936 and irradiation	8	Returned with recurrence after 8 yrs. Died of recurrence 3 yrs. later.
Sept., 1928	70 M	Extrinsic	Adenocarcinoma, grade 3; squamous cell epithelioma, grade 3	Cautery excision, diathermy and irradiation	2	Well 2 years after treatment
Jan., 1929	55 M	Intrinsic	Fibrosarcoma, grade 3	Biopsy and irradiation		Palliation. Probably dead
Aug., 1929	65 M	Extrinsic	Squamous cell epithelioma, grade 3	Cautery excision and diathermy		Died of meningitis 1 month after operation
Dec., 1929	68 M	Extrinsic	Basal cell epithelioma	Diathermy & irradiation	2	Well 2 years after treatment

TABLE 3 — *Continued* *Summary of Results in 38 Cases of Malignant Tumors of the Middle Ear and the Mastoid*

Date of registration	*Age, years, and sex*	*Site of origin of tumor*	*Pathologic Condition*	*Treatment*	*Freedom from recurrence, years*	*Result*
June, 1930	54 M	Extrinsic	Adenocarcinoma, mixed tumor type	Excision and irradiation	8½	Died 10⅓ years after treatment; cause unknown
Oct., 1930	42 M	Intrinsic	Squamous cell epithelioma, grade 1	Exploration and irradiation	Unknown	Patient could not be traced
June, 1931	50 F	Extrinsic	Adenocarcinoma and squamous cell epithelioma, grade 3	Diathermy, radical mastoidectomy and irradiation	10½ yrs.	Well 10½ years
June, 1931	45 F	Intrinsic	Hemangio-endothelioma, grade 2 (angiosarcoma)	Radical mastoidectomy and irradiation	10 yrs.	Well 10 years
March, 1932	63 M	Extrinsic	Basal cell epithelioma	Diathermy, radical mastoidectomy and irradiation	9½ yrs.	Well 9½ years
Sept., 1932	54 F	Extrinsic	Basal cell and squamous cell epithelioma, grade 2	Diathermy		Sequestrum removed at home 4 months later; not traced thereafter
Oct., 1932	52 M	Extrinsic	Basal cell and squamous cell epithelioma, grade 1	Repeated removal with cautery, diathermy and irradiation; gland dissection	1+	Died of recurrence after 8 years
Aug., 1933	47 F	Intrinsic	Squamous cell epithelioma, grade 4	Radical mastoidectomy and irradiation		Palliation. Died of recurrence after 8 months
Feb., 1934	59 M	Extrinsic	Basal cell and squamous epithelioma, grade 1	Diathermy and irradiation	7	Well 7 years
June, 1934	50 F	Extrinsic	Basal cell epithelioma	Diathermy, radical mastoidectomy	7	Well 7 years
Aug., 1936	43 F	Intrinsic	Squamous cell epithelioma, grade 3	Exploration of mastoid and irradiation		Palliation. Died of recurrence after 16 mos.
Nov., 1936	65 M	Extrinsic	Squamous cell epithelioma, grade 2	Cautery excision, diathermy and irradiation		Died of meningitis and septicemia 15th day
Jan., 1937	44 M	Extrinsic	Squamous cell epithelioma, grade 1	Radical mastoidectomy and irradiation		Palliation. Died 4 yrs. later, probably of recurrence

TABLE 3 — *Continued*

Summary of Results in 38 Cases of Malignant Tumors of the Middle Ear and the Mastoid

Date of registration	*Age, years, and sex*	*Site of origin of tumor*	*Pathologic Condition*	*Treatment*	*Freedom from recurrence, years*	*Result*
Feb., 1937	42 F	Intrinsic	Hemangio-endothelioma grade 1 (angiosarcoma)	Exploration, mastoidectomy and irradiation	3½	Well 3½ years later
Oct., 1937	66 F	Intrinsic	Squamous cell epithelioma, grade 2	Biopsy and irradiation	3½	Well 3½ years
April, 1938	59 F	Extrinsic	Basal cell and squamous cell epithelioma, grade 1	Diathermy, radical mastoidectomy and irradiation	3¾	Well 3¾ years
April, 1938	57 F	Intrinsic	Squamous cell epithelioma, grade 3	Radical mastoidectomy, diathermy & irradiation; gland dissection	⅔	Treated elsewhere for recurrence 8 months later
June, 1938	48 F	Extrinsic	Adenocarcinoma (cylindroma)	Diathermy, radical mastoidectomy and irradiation	2½	Well 2½ years
Dec., 1938	46 F	Intrinsic	Hemangio-endothelioma, grade 1, (angiosarcoma)	Removal and irradiation		No recurrence after four years
Oct., 1939	42 F	Intrinsic	Hemangio-endothelioma, grade 1 (angiosarcoma)	Radical mastoidectomy and irradiation	2	Well 2¾ years
Oct., 1939	80 F	Intrinsic	Squamous cell epithelioma, grade 2	Biopsy and irradiation		Probably palliation only. Patient could not be traced
Nov., 1939	27 M	Intrinsic	Hemangio-endothelioma, grade 1 (angiosarcoma)	Exploration, mastoidectomy and irradiation	1⅓	No recurrence when patient was observed last, 1⅓ years later
Aug., 1941	37 M	Intrinsic	Hemangio-endothelioma, grade 1 (angiosarcoma)	Exploration, mastoidectomy, and irradiation		No recurrence after 13 months
Sept., 1941	33 F	Intrinsic	Hemangio-endothelioma, grade 1 (angiosarcoma)	Radical mastoidectomy and irradiation		No recurrence after 9 months
Dec., 1941	3 F	Intrinsic	Neurofibrosarcoma, grade 2	Radical mastoidectomy, diathermy and irradiation		Died 3 months after operation

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DISCUSSION

A. C. Furstenberg, M.D., Ann Arbor, Michigan: Doctor Figi has dealt with this subject in such a comprehensive manner that there is little I can add to the discussion. Moreover, our experiences with this condition are so meager that few of us have been able to formulate rules and regulations for the care of patients with malignant disease of the middle ear.

He has spoken about the etiology of malignancy of the middle ear and mastoid and has emphasized some of the possible extrinsic factors, such as external dermatitis of the auditory canal or chronic purulent discharge for a period of years. Some of these factors, perhaps, are important but it is extraordinary how many individuals we see with chronic otorrhea of many years' duration, and yet, how rarely we observe malignancy in these cases.

There is one other group of etiological influences, perhaps, that might be emphasized at this time, namely, the intrinsic factors. A few of Doctor Figi's cases were proved to be mixed tumors, and a lesion of this type is one which is probably teratological in origin. It perhaps represents some irregularity in the mechanics of embryonic development: a collection of aberrant or superfluous cells, occurring in some part of the body, which do not reach adult characteristics and finally grow to produce what is termed a mixed tumor.

These lesions are most vicious in their clinical manifestations in that they often grow slowly and insidiously for many years. Long after what appeared to be a successful operation for their removal, they recur with increasing malignancy. With each recurrence they depart further and further from their original benign state and become more and more malignant. It is often said that the fate of the patient rests in the hands of the first surgeon. If he can get all the lesion out at the time of the initial operation, there is little chance of a troublesome recurrence. This can frequently be done when the tissue is soft, but in bone it is a very difficult problem. I believe that surgical interference in the case of most of these lesions is futile. Recurrence may not be observed within the first 2 or 3 years, but observations made 6 or 7 years after surgery may be indicative. The percentage of recurrences I believe will be found to be high.

I am glad, however, that Doctor Figi has given us some cause for optimism. He has a marvelous series of cases. Some of the patients I am sure have been cured. His principles of treatment are those which I think all of us may subscribe to at this time, namely, radical excision of the lesion; a technic that will leave the wound open to inspection for the remainder of the patient's life by producing a large external auditory meatus; destruction with electrocoagulation or cauterization, and, finally, intensive irradiation.

I want to thank Doctor Figi for giving us such a comprehensive picture of this condition.

L. A. Schall, M.D., Boston, Mass.: This paper by Doctors Figi and Hempstead is a report not only of the largest series of cases of cancer of the middle ear and mastoid but, in my opinion, it is the best article that has ever been written on the subject. I am in accord with every statement they make, and I confess that I am in accord, perhaps, because they agree with some statements that I made, not so well nor so clear, in a paper that I wrote for the American Medical Association 7 years ago. Cancer of the middle ear is not

a medical curiosity, and it is not a hopeless condition, as Doctor Figi so aptly shows. It is not a hopeless condition if it is recognized early, and if the surgery and radiation are adequate.

I realize that the authors have not used the word "cure"; neither do I use it. I feel that "years of survival" is a better term than cure, in speaking of cancer.

In the discussion of the paper which I gave 7 years ago, Dr. Joseph Beck asked or made a very pertinent statement. I am sorry that he is not here now because I would like to answer his statement. He said, "I should like to know whether these patients are alive five years from now."

My paper was based on 6 cases, with 5 survivals from 2 to 4 years. At that time I showed this slide.

(Slide) Of the 6 patients, one died within 2 years from pneumonia, without recurrence; one died within 3 months from the operation as a result of extension, and the rest were living with no evidence of recurrence.

(Slide) A check-up shows that the patient in whom hemangio-endothelioma was diagnosed is living 12 years after treatment. One patient cannot be traced, but presumably is dead. The other 2 patients were dead within 6 months from the time I read my paper.

Since 1935 I have seen 5 other patients suffering from this disease and a sixth case, a patient of Doctor Lurie, concerning whom I was consulted.

(Slide) Of these 6 cases, but 2 of the patients are living and do not have a recurrence, but the time is too short — merely from 1 to 2 years.

So I would like to paraphrase Doctor Beck, glorious as the results of Doctor Figi's are, and say I would like to see a report 5 years from now because most of his patients have gone only 2 years since operation. I can do no better than re-emphasize the points which he so well emphasized: Cancer of the middle ear and mastoid is not a medical curiosity. It should be recognized early. The treatment should be adequate.

H. I. Laff, M.D., Denver, Colorado: Having seen only 2 such cases, I hesitate to discuss this paper, but certain features about one of the cases which I recently saw may throw some interesting sidelights on this very able presentation.

I am referring to a case, a woman 44 years of age who had been under the care of a competent otologist in another city for a period of 3½ months. At the time he first saw her there was discharge from the right middle ear of 5 weeks' duration, zygomatic swelling and symptoms typical of Gradenigo's syndrome and petrositis — that is, severe retro-orbital pain and abducens paralysis. The roentgenogram revealed mastoiditis and petrositis. He operated on the mastoid and also entered the petrous pyramid.

In spite of the administration of adequate doses of sulfanilamide and sulfathiazole, as well as blood transfusions, the patient's condition did not improve.

In June, 1941, she came to the Colorado General Hospital with symptoms very similar to those already described, and in addition had a right sided facial paralysis. Fistulous openings were found in the nasopharynx, soft palate and right side of the neck. Blood and roentgenographic examinations revealed nothing further, so I concluded we were dealing with a case of osteomyelitis of the right petrous pyramid, complicated by fistulous extensions into the pharynx and neck.

On opening the mastoid, I found that it and the petrous pyramid were filled with profusely bleeding granulations. The dura of the middle fossa lay exposed. I was able to pass probes from the base of the pyramid into the abscesses in the soft palate, nasopharynx and the neck. Still under the impression that we were dealing with petrositis, I passed drainage tubes into these areas. I will show you on slides the position of these tubes. The patient died 18 days after surgery, and at necropsy squamous cell carcinoma was found to be present.

(Slide) This is a picture of the patient which shows the drainage areas on the outside of the neck, as well as the facial paralysis.

(Slide) Here I had the resident pass some tonsil wire into each of the tubes that had been inserted into the fistulous tracts.

(Slide) This is another view of the same procedure.

(Slide) This is a picture taken after the patient died to show that there was extension into the sphenoid so that probes could be passed from the nose into the cranial cavity.

(Slide) Here is a microscopic slide of the granulations, showing squamous cell carcinoma.

Frankly, those of us who had seen this patient did not seriously entertain the diagnosis of carcinoma of the ear. We were so impressed with the diagnosis of petrositis that we failed to think of it. Also, had the granulations removed at operation been subjected

to microscopic study, which is something that we usually do but which for some reason was not done in this case, the diagnosis would of course have been made before the patient died.

Needless to say, I am now having granulations removed from the middle ear examined much more frequently.

HENRY L. WILLIAMS, M.D., Rochester, Minnesota: Members of the Section of Otolaryngology and Rhinology at the Mayo Clinic have worked as admiring assistants to Doctor Figi's work rather than as true collaborators. Doctor Figi has surprised us by his results in the treatment of a condition previously considered hopeless. It is evident from this study that if a malignant lesion within the ear is diagnosed early, better results may be expected. I wish to appear here merely to congratulate him on his work.

FREDERICK A. FIGI, M.D., Rochester, Minnesota: I wish to thank the members for their generous discussions.

Replying to Doctor Schall I might state that 10 of the patients in this group have lived 7 years or more following treatment and 3 lived more than 10 years. A review of the results after another 5 years have elapsed will be interesting, however.

In Memoriam

MAJOR HARRY BAYLISS STOKES, M.C., U.S.A.

1904-1943

Official notification has been received to the effect that Major Harry B. Stokes was killed in action on January 2, while on duty in Africa. Details regarding his death have not been received.

Doctor Stokes was born in India 38 years ago, and he moved to London, England, with his parents a few years later. There he received his early education, coming to the United States at the age of 20. He matriculated at the University of Michigan Medical School, from which he was graduated in 1929. Following internship, Doctor Stokes did special work in otolaryngology at Bellevue Hospital, New York City, and was certificated by the American Board of Otolaryngology in 1933. He practiced in Rochester, New York, and in Tucson, Arizona, for short periods of time, moving to Omaha in 1936. For the past six years he has been an associate in the office of Drs. Gifford, Stokes and Stokes.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the Omaha Mid-West Clinical Society and state and local medical associations. He has had a very active practice, in the midst of which he found time to carry through his assignments as Instructor in Otolaryngology at the University of Nebraska College of Medicine.

He is survived by his wife, two daughters, Susan and Shelley, and one brother, Dr. William H. Stokes, of Omaha.

In Memoriam

James Craig Strong ..... Leadville, Colorado
October 14, 1942

J. Brown Loring ..... Chicago, Illinois
November 6, 1942

John Daniel Fouts ..... Dayton, Ohio
November 10, 1942

Ewing Wilber Day ..... Pittsburgh, Pennsylvania
November 24, 1942

Walter J. Sener ..... Media, Pennsylvania
December 1, 1942

Howard T. Robinson ..... Cumberland, Maryland
December 3, 1942

A. Noah Schiller ..... New York, New York
December 17, 1942

Frederick C. Simon ..... St. Louis, Missouri
December 31, 1942

E. F. Stroud ..... Corpus Christi, Texas
December 31, 1942

Harry B. Stokes ..... Omaha, Nebraska
January 2, 1943

J. F. Duane ..... Peoria, Illinois
January 21, 1943

TRANSACTIONS
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Editorial

The President's Message

As this new and somewhat critical year of 1943 registers the further history of our splendid Academy, the officers extend sincere greetings to its representatives throughout the country, leaders in the ophthalmic and otolaryngologic specialties. Many new members have been admitted to the Academy who have passed through the baptismal fire of the searching Board examinations, approved as to ethics, personality and technical ability, thus to be added to the published Academy lists throughout this country, an indelible stamp of worth.

The annual gathering has become a great forum for exchange of ideas and experience, for the contribution of newer bits of knowledge, for refreshing acquaintance and knitting in closer friendship the leading spirits in these great specialties.

We all are agreed that for this particular year great emphasis should be put upon teaching — teaching in the scientific session, instructional course and laboratory course, with but a modicum of emphasis upon the social features which has always meant so much to us. This instruction should as far as possible contribute to the war effort in adding skill and understanding to those in the armed services and those in civilian practice of medicine.

Your Council Program Committee has made a faithful effort to arrange such a program in comprehensive detail with particular emphasis upon war medicine, war injuries and war prophylaxis. The Academy should also make every effort to extend generously its fellowship and opportunities to the representatives of ophthalmology and otolaryngology in South America.

This particular year is fraught with a peculiar anxiety—the seriousness of our country's situation. To be socially entertained is not the keynote; the happy association with fine, spirited men will continue, and hundreds of you as heretofore will return to review the Academy picture in your own homes. Make your Academy contribution this year a complete and stimulating cooperation.

Most cordially,

JAMES A. BABBITT

A PANEL DISCUSSION ON INDUSTRIAL OPHTHALMOLOGY

Those participating in this panel were the following members of the Joint Committee on Industrial Ophthalmology, Section on Ophthalmology of the American Medical Association and the American Academy of Ophthalmology and Otolaryngology:

Albert C. Snell, M.D., Chairman, Rochester, N. Y.
Hedwig S. Kuhn, M.D., Hammond, Ind.
Thomas D. Allen, M.D., Chicago, Illinois
John B. Hitz, M.D., Milwaukee, Wisconsin

And by Invitation:

Joseph Tiffin, Ph.D., Purdue University, Department of Applied Psychology, Lafayette, Indiana

Mr. Thomas Lane, President of Indiana State Personnel and Management Organization, Hammond, Indiana

Mr. Cliff Boughner, Safety Engineer of Carnegie Corporation, Gary, Indiana

Adolph G. Kammer, M.D., Medical Director of Inland Steel Corporation, East Chicago, Indiana

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CHAIRMAN SNELL: Gentlemen, your Committee on Industrial Ophthalmology has been working for a year, trying to solve the problems concerning industrial ophthalmology by getting information first-hand. We hope that you who are present will contribute to this information.

The panel is made up of about an equal number of doctors and others who are interested in industry from the standpoint of management and safety. I am not going to take up your time by making any remarks on the subject which I have outlined in the syllabus. A more realistic service in ophthalmology has been pointed out. Mr. Tolman, consulting engineer of the National Society for the Prevention of Blindness, has made a careful survey of 50 industrial plants, showing the general lack of adequate ophthalmic service in industry. I think all of you who have had any experience in industrial work, especially that which relates to vision, must be impressed with the fact that there are many gaps in the field of this service.

Recent statistics, relating to defective vision among employees, show that an average of 15 to 35 per cent of them have defective vision. This is a definite handicap to production, and it is only one example of the need for a more realistic ophthalmic service for industry.

We felt that the best way to proceed with this program was to have Professor Tiffin speak first. He will give us a statistical background for this panel discussion. Professor Tiffin.

PROFESSOR JOSEPH TIFFIN: I speak entirely from the point of view of the layman and from the point of view of a man in a university who is attempting to work out for industry a set of procedures for employee placement, transfer, allocation and possible selection, which will be of some help to them in today's job of putting a large force of men and women, more women as time goes on, in a vast variety of jobs which are in many respects new ones — new as far as the activities of the present employers are concerned.

I think it would be worthwhile to give a very brief picture of how Purdue University's interest grew in this branch of vision research in order to show the relationship in the field, as we see it, (1) from the point of view of the layman and the technical university, (2) the industry — the industrial man, the industrial relationships man and management in general, and (3) the professional men who deal with eye problems.

Our problem has been to work out tests of various types for the allocation of employees according to their fitness and according to the job requirements. To show the relation of the types of testing to the vision program I might illustrate as follows: There is a demand for machinists and machine operators. With classes in defense training it very often happens that a man will come into a plant and say, "I would like a job as a machinist."

"Have you ever been a machinist?"

"Yes, I took a six weeks' course at the training school."

A panel discussion on industrial ophthalmology was held in connection with the Forty-Seventh Annual Convention of the American Academy of Ophthalmology and Otolaryngology, at the Palmer House, Chicago, Illinois, October 12, 1942, Dr. Albert C. Snell presiding.

Since machinists are needed very badly, he is very likely to be hired. Now the question is, is he a qualified machinist or not, and if so, what things does he know about a machine shop? The test deals with everything concerned with a machine shop. From the score a breakdown is made to show exactly not only how much about a machine shop such a person knows, but also how much he knows about a grinder, bridge work, planers, lathes, milling machines and the total machine shop operation. That sort of information is a great help to industrial management in properly placing the employees, particularly when, as occurs today, one deals with individuals who are not well grounded in all skills, but who have had a very hurried type of training which will fit them for one job if that one job can be ascertained.

Now from the visual point of view, the statistical work that we have been carrying on represents an analysis of around 9,000 visual records obtained in one survey that was directed by Dr. Hedwig S. Kuhn in a mill in the Calumet region. In this particular survey, we obtained comprehensive data concerning every one of those 9,000 employees, namely, with regard to accidents, production, merit rating, absences for two successive years, minor accidents, lost time accidents and all the other personnel data that were obtainable, such as age, sex, marital status and so on.

We checked and correlated the results of the vision tests obtained in that survey against various indications of employee performance on the job; that is, production and accuracy, absenteeism and so on. We correlated the results of the vision tests against such factors as age, experience on the job and so forth. We made a statistical analysis of the average pattern of visual performance among employees on different jobs or in different job classifications or different job groups. Some rather interesting things were brought out. In one particular analysis we very carefully compared the visual characteristics of the following groups of men: foremen, clerks, electricians, machinists, cranemen, hookers and laborers, who represented job types in which there was a minimum of 250 employees per job, and in which there was a maximum of 710 employees per job in the case of laborers.

Interestingly enough, the visual pattern for these different jobs did not agree from one job to another. In many cases we found great differences, and statistically significant differences. For example, the foremen excelled statistically in tests of depth perception, color vision and in near point acuity. They did not differ significantly from the plant average in color vision, in phoria near or in acuity near and again did not differ significantly from the plant average in the other characteristics. The electricians were significantly better in distance acuity, but, strangely enough, were significantly poorer than the plant average in acuity of the worst eye at distance.

To take a group at the opposite extreme, the vision of laborers differed significantly from that shown to be the plant average in only two respects, namely, in depth perception and in near point acuity; and in both of these tests they were significantly poorer than the plant average, and they were significantly better than the plant average in no respect.

The datum which the employment man can use very definitely in many types of employment is simply this: Among the different types of jobs in his particular plant he should recognize the necessary pattern of visual characteristics (visual performance scores) essential for different jobs. He will almost certainly find, as we found in this particular survey, that it is not desirable to attempt a single, more or less arbitrarily determined, standard for every employee. That is not desirable with the labor market as it is today, since one cannot get enough people whose visual qualifications come up to high standards.

Another reason is that even though the labor market were such that it would be possible to select and to employ according to a more or less arbitrarily determined standard, all the statistics indicate that from the production viewpoint this would not be desirable, for the reason that the same visual pattern for one job is not necessarily characteristic of high production in some other job.

The functioning of a placement program, from the point of view of management, personnel, industrial relations, safety man and other men in the management group, depends upon a number of factors. First, there must be some sort of instrumentation for testing vision which is simple and is not professed to be medical. It should be clearly so indicated to the non-medical persons who use it in the

industrial relations office. However, the use of such instrument should produce test results of a type that will be meaningful to the professional man, but which in no sense attempts or makes it possible to replace the consultation or the referral of the data to the ophthalmologist. In other words, the instrumentation will best accomplish its purpose if it is such that it indicates what visual performance characteristics the employee has, such information being used by the employing man, in placement. But at the same time it should serve the additional purpose of giving the professional men, who are associated with the plant activities, information about these performance data which they may reinterpret and which may give them a clue as to that particular employee's eyes with relation to his particular job. This is illustrated by some of our data. We found a rather interesting and striking statistical relationship, a negative relationship between distance acuity, as measured quickly by a simple, nonmedical test, and production at a very near point.

To illustrate this point with a specific example: On a particular job the employees with a distance acuity of 20/80 were producing on the average four dozen hose per hour; the employees of equal experience on the same job with a distance acuity of 20/20 were producing about three and four-tenths dozen per hour; and a few employees with a distance acuity close to 20/10 were producing only two and one-half dozen per hour. These data certainly open a question as to whether the distance acuity measurements obtained instrumentally by laymen would be the same as the distance acuity measurements obtained by a professional man in an office. However, when the employment man finds any test, whether it logically makes sense or does not, by which he can show statistically that the employees who score thus and so will produce two and one-half dozen per hour whereas the employees who score differently will produce four dozen per hour, he has something that he is going to use in his placement of employees. He cannot escape using it, because a vital part of his whole job is to look for methods and devices which will enable him to spot the people who will do the best productive job. From the employment man's point of view, visual tests simply serve as a means for better placement of employees according to the job.

It would be very foolish for us to say that since there is this negative relationship between distance acuity, as functionally and quickly measured, and production, we should employ for close work only those who have subnormal distance acuity. But it would be just as foolish to employ for close work only those who pass the normal distance acuity test.

Further testing, in which we changed the distances and tried acuity at closer distances, revealed that one can get a positive relationship between such an acuity, determined at approximately the work distance, and production. This should be recommended to the employment man as the better procedure for placement. I mention these relationships as an illustration of the general point of view that is taken by the employment man in ferreting out from among potential employees for this, that or the other job those whose particular patterns of skills, trade knowledge and so forth will best suit them for that particular work.

I mention these specific examples because they show very definitely that the pattern of visual skills necessary for success on a specific job is not the same as the pattern of visual skills necessary for success on some other job. When one has a pattern of visual skills which makes for success in a specific kind of a job, he has a measure that is as accurate a measuring stick as a ruler from the Bureau of Standards Office. Production measured week after week correlated with visual data will demonstrate the dependability of visual testing data.

From these data, in which we have given about every type of vision test that could conceivably be developed for a layman's use, which tests were given to approximately 9,000 employees in one plant and a total of around 9,000 or more in several other plants, making a total of nearly 18,000, we have attempted to put together the particular types of tests that, on the average, would seem to be most adapted to the largest variety of jobs. We have worked out entirely new distance acuity tests and tests adapted to the near point, so that the visual angle at distance and at near is the same, but obviously the size is changed so as to make the test effective at both distances.

An instrument has been designed for non-professional use. I don't think there is a gambler's chance of anybody ever being able to change that instrument so that a refraction could even be guessed at from the results obtainable with it, and we have deliberately avoided making it possible to do that. With the particular technique, we have worked out a system of recording which is a graphic system on the one hand and a punchcard system on the other — a punchcard system, however, that does not require expensive machines for sorting. The sorting is done with a knitting needle; so that an employment man, once he has a stack of these record cards, can in a few minutes sort out from his group of employees for example, (a) those who are visually adapted for certain types of work; (b) those who probably would be visually adaptable if they obtained proper professional service; and (c) those who, for some reason, are not properly adapted even to the job they are now on and, therefore, arrangements should be made for them to obtain the professional service necessary to fit them for efficient production.

As a high-light, that is a picture of our point of view from the statistical angle. I hope that I have made it clear that we are not in any way attempting to do something here that can better be done by medical men. Our feeling from start to finish is that what we should do is to serve, you might say, as liaison officers between professional men and industry, and work out such methods and technics that will make that relationship closer and give the industries a clearer picture of the services which they have every right to expect from professional men when that relationship is correlated through some such program as we have outlined.

HEDWIG S. KUHN, M.D.: One thing that I wish to say before I begin my assignment entitled "Visual Testing in Industry" is that while Doctor Tiffin stated the new instrument is for non-medical use or non-professional use, he did not mean that it is to be divorced from the medical department or that any of the interpretations of the findings are to be made by the technician who does the test.

In a war program that is being geared to top capacity, we feel that not only must we reduce waste of time, skill, equipment, power, materials and man power, but that, as Doctor Tiffin has shown, we must increase production and accuracy. We feel that it is as useless and unfair to ask an employee to improve his work and reduce his mistakes when he is not able to see his work as it is to ask a man with laryngitis to talk more clearly. A real understanding of all the elements that make for visual skill has only recently been brought to the front, and the protective measures needed to cut down eye hazards, although understood, are really only now beginning to be used. Management problems or safety problems inside the plant heretofore have not been matters for the medical profession to solve, and they make a very new field for the ophthalmologist.

Visual testing in industry falls mainly into three categories: First, pre-employment tests; second, periodic rechecks of special groups, such as crane operators, those employed in dangerous occupations, inspecting accuracy jobs, micrometer reading, etc.; and, third, plant surveys. For the duration of the war, there will be fewer persons rejected in the major war industries and fewer applicants rejected for civilian jobs, but for this very reason — and it has been a big boost to this whole concept — there must be more practical and more accurate admission records in order that job allocation can be based on visual safety and visual skills of the applicants before they are assigned. If one lowers the admission standards considerably, it is desirable to put a man on the job that he can do best.

The personnel or employment director devotes only a limited amount of time to determine that a machinist, who gives a splendid report about his accomplishments and who demands a certain salary or wage, can actually operate that $10,000, $15,000 or $20,000 machine. He refers to the employee's record, and he checks on his experience, etc. In the same way, in very much less time in minutes actually, he can judge the applicant's skill from a physical standpoint, especially where this involves vision. In operating many of the important machines in war industries, vision is an essential factor. As the young man is replaced by the man over 40, vision becomes a more and more important issue in performance of these skills. We want to conserve eyes and production efficiency. In certain dangerous jobs, which are especially dependent on

full visual acuity and coordination, periodic rechecks are necessary to discover the changes in vision. Pilots are receiving such rechecks. They are certainly exceedingly important in this and in other vital jobs which require precision instruments, instrument reading and the inspection of blue prints, where accuracy is essential. Precision is altered by changes which occur within the eye. In these types of employment, rechecks are indicated periodically. Some industries have unexpectedly been made a part of the vital war effort, but have never had any plant surveys made or visual testing programs instituted. Divisions and sections of these plants, where the work to be performed demands critical vision, will profit tremendously by a visual survey geared to meet their individual needs.

One common error made by the doctor is that he wants a fixed, inflexible program; but industry demands a different program. Industry has specific problems to be solved in the order of their importance; and unless doctors help industry with their most urgent problems, there will be no progress in any visual testing program or rehabilitation.

The basic visual data that industry needs to have recorded are the following: Distance and near acuity without and with glasses, muscle imbalances (phorias) for distance and near, color appreciation levels (not just whether color blindness is present or not), depth perception; and in addition to these, where especially indicated, test data concerning near vision, based on the individual's needs and that of the job. Also, the data should contain certain tests for age groups. Visual functions indicated in these data are elements of visual skills.

Tests which correlate the above data are not clinical tests of physiologic function nor do they take into consideration pathology which may be present. Such information should follow these preliminary tests for each employee who registers a serious visual defect. For example, when an ophthalmologist finds a visual acuity of 20/100 in one or both eyes, he should find out why, and correct it, if possible.

The wide demands on vision in jobs such as that of a crane operator who is dependent in a large measure on stereopsis at 150 feet and that of a looper in a hosiery mill doing piece work 8 inches distant from her eyes reveal the prime necessity of classifying visual skills or aptitudes along with mental and physical skills in relation to specific jobs. Especially are these near-point occupations, which prevent accurate perceptual power, a drain on any employee's fatigue reserve. I wish to place special emphasis, therefore, on the necessity for near vision tests where the employee is 40 years old and over, or where the visual performance at working distance is within the normal reading distance. Therefore, applicants for jobs that require visual performance at a working distance within 16 inches need added standardized tests for near-point convergence and near-point accommodation and the refraction tests at the work distance with correction for this working distance. The ophthalmologist is a positive obstruction to the war effort who, knowing that the patient is an industrial employee, makes a refraction and prescribes streetwear glasses without considering the patient's work distance and also without finding out for sure that the patient's visual performance is adequate at that distance.

The ordinary bifocal lens for distance very often does not serve the purpose of a man when he is at work; both the type of spectacle and the working distance must be considered. One can give him the so-called occupational lenses just as one may prescribe occupational shoes or gloves, which he wears while at his machine for protection and efficiency.

The question of minimum visual standards for specific jobs is under consideration. A job classification needs to be relatively simple and elastic and appliable to industry anywhere. Medicine can conserve not only the health of an individual's eyes, but it can help to increase production schedules which is an issue that is becoming more and more important.

The success or failure of an industrial visual program depends on our understanding of the visual requirements of industry, the need of accurate recorded visual data which are necessary for proper placement of workers and a plan for detecting and correcting visual defects.

CHAIRMAN SNELL: The Americen College of Surgeons has set up an organization to study adequate medical staffs in industry, based on certain principles. They found from surveying a large number of plants that there are very few industrial plants in the entire United States and

Canada that can meet the requirements which they have laid down, and that only a few large plants in the United States employ a full-time ophthalmologist. Thus, it seemed to us it might be instructive to have someone present the topic which Doctor Hitz will now discuss; that is, "The Industrial Medical Staff."

JOHN B. HITZ, M.D.: When I first consented to be on this program I was doing a fair amount of industrial ophthalmology. I am now doing considerably more, but the present set-up does not seem to have the politico-economic complication with which private practice becomes involved. The relationship of medicine, including ophthalmology, to industry has become inextricably bound up with the problems of socialized medicine, the expansion of unionism and the increased control of government over business. Certain principles I believe can be set down, however.

Complete medical supervision of the employee in any industry is in the public interest. This should include pre-employment examinations, periodic check-ups, allocation of employees to jobs which they are physically capable of handling, treatment of all accidents and diseases contracted in line of duty, emergency management or treatment of all other conditions and supervision and consultation in all matters concerning industrial health and safety.

The ophthalmologist should definitely be a part of this picture. The specialist in industrial medicine cannot possible be expected to handle problems of ocular safety and ocular fatigue. The problems of proper lighting and visual protective equipment should probably properly be in the hands of lighting and safety engineers, but I am sure in many cases much benefit would result from consultation with an industrially-minded ophthalmologist. Personnel officers could certainly benefit by having some knowledge of prospective employees' visual capabilities or handicaps.

The medico-legal value of having recorded visual acuity records on each employee at the beginning of his employment seems too obvious to need discussion here. It should not be difficult to convince intelligent union leaders of the fact that pre-accident visual records work to the advantage of both the injured employee and the management. I was reminded last night that the state laws, as far as compensation is concerned, vary considerably, and in a good many states the pre-employment vision, or the vision prior to the injury, is not considered as a basis of compensation. However, it is in some states, and it would seem logical to believe that a known degree of vision prior to accident will become more and more an accepted factor in computing compensation.

Part of any complete medical service should include periodic ophthalmological examinations. Under ideal circumstances this work should be done by the ophthalmologist, himself. At the present moment, however, we are all struggling to be eminently practical and desire to accomplish the most with the least loss of time.

The problem would seem from the ophthalmologist's standpoint to resolve itself into two questions: First, how can this work best be done, with due consideration given to conservation of the ophthalmologist's time and, second, by what method should the ophthalmologist be compensated for his service.

The obvious answer to the first question is in the use of trained technicians who can make screening visual examinations. I hope that we have passed the point where the testing of distance acuity with the Snellen chart, often very poorly lighted, is considered adequate as a screening examination. As a minimum, an adequate screening test should be a test of distance and near acuity, a test for muscle balance, and in certain jobs, tests for color vision and depth perception. There are several batteries of tests which can be handled by intelligent technicians with a few weeks' training. In fact, even the complicated visual tests in use in the Army Air Corps today are being conducted primarily by enlisted men. Often these men have had a short period of training.

Larger plants, of course, can employ full-time technicians. In smaller plants the industrial nurses may be assigned to give visual screening tests, although this may not be ideal. The cooperative employment of a visual testing technician by plants having under 1,000 employees is well within the bounds of practicability. This could be arranged through county medical societies, the visiting nurses associations or local ophthalmological groups.

There has been considerable criticism in some parts of the country to the training of non-medical personnel as visual

testing technicians. This has come usually from individual ophthalmologists, or, surprisingly enough, from medical societies, both state and local. The usual complaint is that too many medical services have already been taken over by technicians — that industrial nurses are already practicing medicine of a sort in too many places. My answer to that is that 75 per cent of the visual testing and refracting is already being done by non-medical persons.

The answer to the question of compensating the ophthalmologist should not present much difficulty at this time. In the first place, the war and its taxation program has reduced the physician's desire to over-accumulate wealth and, second, at least so I am told, some industrial organizations are happy to find legitimate ways of reducing their tax burden. However, the question of who should make the examinations and how these and necessary appliances are to be paid for is not settled.

No industrial visual efficiency plan can function satisfactorily unless the management is made to see the economic value of such a plan and is willing to carry at least part of the financial burden. One of the usual methods is for the employer to pay the cost of the examination, and the employee to purchase his glasses at contracted prices. Undoubtedly, there are many other ways by which this problem can be worked out, but from my experience unless management is willing to carry part of the cost, no visual program can be made effective. If management can be convinced of the economic value of having its employees visually efficient, adequately protected and in jobs for which they are visually adapted, there should be little difficulty in settling on methods of compensation for the ophthalmologist.

Most industries employing 20,000 men or more can well afford a full-time ophthalmologist, if they can find one. Smaller industries will prefer to employ part-time oculists either on a salary basis or on the basis whereby refraction examinations, treatment and consultations are paid for on an individual fee basis.

Incidentally, I believe it behooves us as ophthalmologists to educate ourselves a little further in the line of industrial requirements. Doctor Kuhn has emphasized the necessity for considering the individual job that the employee does before attempting to fit him with glasses. Furthermore, it certainly would do no harm for the ophthalmologist to educate himself in problems of lighting, the use of color in lighting and about various safety devices. Lighting engineers have done an excellent job in recent years, but the final answer to proper lighting has not yet been written, as demonstrated by the great numbers of employees still coming to the ophthalmologist's office with complaints of eye strain due to inadequate lighting.

An interesting new development has been the use of contrast colors on machine parts to promote both efficiency and safety. The engineer can work these problems out, but the oculist who listens patiently to the complaints of the employees is in a position to give valuable help and advice.

Present safety devices, to my mind, leave much room for improvement. For example, no one has solved the problem of preventing fogging of cup goggles. The ophthalmologist is in a position to help the engineer only if he has knowledge of the mechanical phases of the problem as well as of the medical.

To conclude, my firm conviction is that the industrially-minded ophthalmologist should cooperate more fully with management in setting up visual efficiency and safety programs, including the following points:

1. Periodic visual surveys, that is, screening tests.
2. Adequate refraction service to those employees needing corrections.
3. Consultation with directors of personnel on methods of allocation of jobs to correspond with the employees' visual capabilities.
4. Consultation with lighting and safety engineers in the capacity of the man who has to hear the complaints from the employees themselves.

CHAIRMAN SNELL: You will notice from the synopsis that we did not provide very much time to discuss the ordinary injuries that follow as a result of flying foreign bodies becoming lodged in an eye. We felt that all of you who do industrial ophthalmology would be familiar with those problems; but in the field of injuries other than those that occur from foreign bodies, we have much to learn. We are very happy, indeed, that Dr. Thomas Allen is willing to give us the result of his experience and knowledge in this field.

THOMAS D. ALLEN, M.D.: We are accustomed to think of evolution in terms of millions of years, but in the last 40 or 50 years there has been a very evident evolutionary tendency in industrial medicine and surgery under our very eyes, and we, you and I, have helped to accelerate the process.

Until comparatively recently, people as a whole, and the medical profession, too, were more interested in gross defects of obvious etiology, obviously requiring certain dramatic treatment. The lesser ills, although they may have been almost as disabling, were considered casually if at all. But as refinements have been the result of our conscious cooperation and the rivalry of thoughtful minds, more and more interest has developed in these lesser ills. Sometimes we may think that the interest goes too far, as with our modern vitamin therapy for whatever one thinks may ail one, but in general we are pushing back the boundaries of ignorance.

Our present subject indicates the continuation of this same evolutionary tendency. In fact, the subject has become so enormous, and is growing greater with the expansion of industry, that it is impossible more than to touch on the most salient features.

A. Injuries from gases and fine particles that act by direct contact and by inhalation:

Many industries have problems of this kind which usually can be adequately controlled by the installation of proper ventilation.

A specific illustration is found in an article by David Rankins of Liverpool, England, in the British Medical Journal of July 4, 1936, pp. 6-9. In 1927, a small factory, which manufactured artificial silk, was started which got into production on a large scale in 1928, and during the two years 1928-9 1,598 cases of keratitis occurred among the employees. The symptoms were blurring of vision, halos about lights, pain, lacrimation and blepharospasm. Some cases were mild; others, quite severe. About two-thirds of the employees seemed to be immune. The attacks lasted 3 to 5 days following which recovery was complete. The findings were: Marked conjunctival redness especially in the interpalpebral fissure zone; occasional punctate hemorrhages; ciliary injection; fine punctate epithelial elevations followed by desquamation of considerable corneal epithelium especially in the central portion; not iritis; often severe blepharospasm. The patient's breath would have the odor of onions. There was rapid and complete repair. In mild cases the epithelium was not desquamated. The condition followed not a single exposure, but repeated daily exposures over an 8 hour day, and would come on after a latent period of 6-8 hours (i.e. it was remarkably like a photo-ophthalmia).

The author described in brief the essential steps in the factory for the manufacture of artificial silk from wood pulp. (1) Soaking in strong caustic soda, (2) macerating in grinding machines, (3) treating with CS_2 to produce cellulose xanthate (4) treating with lye (18 percent), (5) dissolving in 4 per cent lye, (6) forcing through fine nozzles into a 10 percent solution of H_2SO_4 where it is coagulated, (7) guiding the fine coagulated threads over a pulley to shake off adherent solutions and then (8) winding in a spinning box to form "cakes." "Cake changers" place the "cakes" in ovens to dry. The spinners and the cake changers were equally affected. The spinners had more frequent "viscous splash" accidents, but different types of lesions (usually of the skin) were present in these cases — immediate and temporary irritation. (A similar condition was reported from a factory in Strasburg, said to be perfectly ventilated.) A complete overhauling of the ventilation system whereby the exhaust was all **drawn off at the floor level** produced an immediate and dramatic lessening of the incidence of keratitis. One relapse was experienced when as a result of a coal shortage the fans over the acid tanks were stopped, although the ventilation otherwise was continued as usual. At this time it was found that thioformaldehyde, a gas, was formed from the CS_2 in the presence of nascent H. This had an odor like onions, and it was thought that it may act by inhalation, predisposing certain individuals to the abiotic-like action of the gases on the epithelium.

I have given this example in considerable detail to illustrate the fact that some injuries do not appear at once but after a latent period, although the injury is, in part at least, a contact one. R. McDonald (Bulletin 46, Occupational Diseases, Prevention Division of Commonwealth of Pennsylvania, Dept. of Labor and Industry, 1938, p. 38) seemed to be more impressed with the actual contact of drop-

lets of CS_2 and H_2S in his account of the ophthalmological aspects of the hazards in the rayon industry. Both he and Dr. M. Davidson (Archives of Ophthalmology, April, 1939) believe the keratitis is produced by actual contact of the fine droplets of spray clinging to the cornea. In sufficient concentration and with long enough exposure CS_2 may produce headache, mental confusion, optic atrophy and cerebral degeneration. (Benedict-Digest of Treatment, 3:546-548, Jan., 1940). Dinitro-benzol is a similar poison.

Previous to 1913, wood alcohol was used to clean large beer vats. Charles Baskerville reported on the dangers of blindness from this source.

The dangers of carbon monoxide, especially in closed garages, are well known.

The fumes of molton lead, of chlorine, bromine, hydrochloric acid, diethyl sulphate, ammonia and many other chemicals are very irritating to all mucous membranes. Fine soot has caused a good deal of conjunctivitis in the chimney sweeps of England. Powell has described an interesting type of conjunctivitis from peat dust (Am. J. Ophth. 17:206, March, 1934) in a relatively small area, about 150,000 acres, west of Stockton, California. Apparently the irritation, which was uniocular, came from the acid soil blown about during hot dry spells and from the fine spicules of silica present therein. The irritation was always quickly relieved by cocaine, homatropine and applications of mild silver. The industrial aspect was as follows: The affected persons were mostly white people working on the farms during the dust storms. Dark skinned races and acclimatized people were almost immune. Slightly different was the report by Anneberg of Carroll, Iowa, of a series of 6 cases of keratitis from pigweed and redroot pollen. (Am. J. Ophth. 21:11, November, 1938.)

B. **Fluids:**

Splash accidents have been eliminated in great measure by the wearing of hoods, proper clothing, gloves and masks for face or head; but fluids containing poisonous chemicals still cling to the hands of careless workmen, and girls painting ceramics still moisten their brushes between their lips and are thereby accumulating lead in their systems. Lead is known as the greatest of our industrial poisons, as it is used so extensively in manufacturing and in small industries and trades. It may produce widespread disabilities, acting mainly on the nervous system. With the expansion of industry in smaller communities, safety directors must be on guard against many dangers, one of which may be illustrated by reciting in brief a case history. A young lady 16 years of age came to see my chief, Doctor Wilder, some 20 years ago, complaining of poor vision of a few weeks' duration. A diagnosis was made of neuroretinitis. A classical star figure was present at each macula. She was found to have a lead line at the gums and about the finger nail bed, and her urine contained an excessive amount of albumin. She had been drinking water from a cistern fed by lead pipes.

A shoe dye used some years ago by bootblacks caused a syncope and amblyopia; laws were at once passed banning the further use of this dye.

C. **The heavier metals and certain chemicals and drugs:**

Many arsenical, mercurial and silver compounds, lead, quinine, cocaine, amyl nitrite, benzol, carbon tetrachloride, methyl alcohol and many other drugs are being used in increasing quantities by industrialists who are not always acquainted with the dangers inherent in their use. (Pelner and Saskin, J. A. M. A., August 8, 1942.) Thallium, chromium, dinitrophenol and many coal tar products also should be mentioned. The Department of Labor has published excellent lists and resumes of dangerous chemicals and the trades in which they are used; also methods of handling them and of guarding against poisoning. (See especially Occupational Hazards and Diagnostic Signs, U. S. Dept. Labor, Div. of Labor Standards, Bull. No. 41, 1942.)

D. **Infections:**

As physicians, we must be most careful of the medication we, our assistants and nurses use in our patients' eyes. There are extraordinarily few cases of infection reported from "drops" of one kind or another, but this is a possibility that we must recognize. A number of workmen had been apparently affected by contamination from a bottle of fluorescein before the source was found. Frequent boiling of the solutions end this source of contagion. A 2 per cent solution of mercurochrome is almost as good a staining reagent as any other, and is itself moderately antiseptic. Almost all medications used in the eyes may become affected if the

droppers are allowed to gather dust and germs by touching fingers, lids, the table or the outside of the bottle. (Mundt's Contrib. J. A. M. A., July-August, 1942.) For that reason most "eye drop medicine" is put up in small "dropper bottles" which contain only enough for a few weeks' use at the most. Also, we must guide our patients to druggists whom we know to be "infection conscious." A slovenly druggist is a great danger to a community.

Infection of a luetic nature was passed to a young boy by a fellow workman who removed a cinder from his eye with a handkerchief which the workman whipped out of his pocket, rolled into a pointed sponge and moistened with his saliva. The boy reported six weeks later at which time a chancre was present on the upper lid. A slovenly first aid attendant is a liability rather than an asset. We all know the dangers of the common towel in disseminating trachoma, the dangers of the sneezer, etc.

A recent severe epidemic of keratoconjunctivitis occurred in San Francisco, which was not limited to workers in shipyards. A full description is given in the American Journal of Ophthalmology by Rogan and Crawford, September, 1942. They append a bibliography concerning similar epidemics.

E. **Radiant energy:**

Mr. R. S. Bonsib, an engineer with the Standard Oil Company of New Jersey, has epitomized this subject from an industrial standpoint very well. (Twelfth Annual Safety Convention, Greater New York Safety Council, April, 1941. "Occupational Eye Hazards with Special Reference to Hazards other than from Foreign Bodies and Traumatic Injury.")

"Radiation from an intensely heated solid or vapor may be divided into three portions: (a) invisible infra-red rays, (b) visible light rays, and (c) invisible ultraviolet rays. Exposure of the unprotected eyes to very intense light sources, rich in ultraviolet radiant energy, produces an acute and painful inflammation of the eyes. Long continued exposure or frequent short exposures to ultraviolet rays of even moderate intensity may have a similar effect on the eyes. The disease produced is a form of inflammation of the eyelids commonly called Electric-Ophthalmia or Photo-ophthalmia." (This is about the same as a sunburn of the skin, and like sunburn comes on several hours after exposure.)

"A series of experiments have been made by the United States Bureau of Standards upon various kinds of colored glasses. It was found that most black, amber, green, greenish yellow and red glasses are efficient for protecting the eyes from ultraviolet light. For shielding the eyes from infra-red rays, yellowish green, sage green, gold-plated and bluish-green glasses are the most serviceable although color is no criterion of efficiency. Glasses having a gray or neutral tint are the most agreeable, as they do not alter the color of objects."

Photo-ophthalmia is essentially an abiotic change; it is not a "burn" in the usual sense. There is a latent period of 4-8 hours between the time of exposure and any recognizable subjective or objective change. It is produced in the superficial tissues only because the ultraviolet wave energy does not penetrate deeply. The individual cells, especially in the deeper epithelial layers, swell, their protoplasm undergoes necrolysis, bullae and blebs form, the contents discharge and the base is apt to become infected. Depending on the severity of the exposure, the lesion undergoes rapid or dalayed healing. A "burn" on the other hand is the result of infra-red energy; the effects are an immediate hyperemia, necrolysis or eschar.

X-rays are shorter than the rays usually included in the "ultraviolet" band. Some of these penetrate deeply and cause serious tissue stimulation and death. It is for this reason that it is used in therapy. Cataract formation from ultraviolet light is theoretically impossible; Verhoeff and Bell (Proc. Am. Acad. of Arts & Sc., 1916, 1:630) showed that ultraviolet light does not penetrate as far as the lens. Glass blowers do not have the photo-ophthalmia that ultraviolet light produces. Visible light does not produce pathology in the ordinary intensities of daylight. Infra-red energy does penetrate the eye (eclipse blindness is a heat burn of the macula). There still exists a controversy regarding the exact mode of formation of glass blower's cataract. It's formation takes from several years to 30 years. Other occupations in which this disability is found are: Chain-making, smelting, tin-plate making, gas welding and cutting. The wearing of goggles is essential in these industries.

F. Illumination:

Many years ago miner's nystagmus was thought to be caused by insufficient illumination. Today there is no excuse for such poor lighting in mines as to contribute to nystagmus. (The question of cause and effect never has been properly settled. Do the miners, who have inherited poor vision and nystagmus, remain in the mines while those who have good vision take up other occupations? Or does reduced illumination really contribute to nystagmus?) There has been such a development of lighting that we are having to study some of the bad effects of too much and of improperly placed illumination. Of course, daylight is usually the best general lighting, but as many plants cover whole acres of land and some have to be prepared for continuous work even during blackouts, artificial illumination aided by proper painting of reflecting surfaces must be substituted.

Long, luminous tubes of light should be enclosed in a trough reflecting the light to a bright ceiling. Naked points or lines of light striking the eyes at an angle of 20° or less from the line of sight are particularly annoying and constitute part of what we call "glare." Automobile headlights, especially when reflected from a wet pavement, and thus partly polarized, are particularly annoying and dangerous.

Good general illumination in a factory will reduce the dangers, uncleanliness and disease of the workers; it will keep them happier and tend to promote cleanliness; eye strain, headaches and bad tempers will be lessened, but the millenium is not yet.

CHAIRMAN SNELL: We have been extremely fortunate to have non-professional men as well as physicians on this panel. We hear a great deal about these problems from the medical standpoint, but it is rather unique in the Academy to have men who represent management and personnel to come here and present some of the problems from their point of view. It is with great pleasure that I now introduce Mr. Thomas Lane, who is the President of the Indiana State Personnel and Management Organization. He will speak on the subject, "The Visual Service Which Management of Personnel Departments Considers Adequate."

MR. THOMAS LANE: As the industrial revolution has unfolded, this field of personnel management has developed. It started about 15 or 20 years ago with what we called the "paternalism era." It has had a great impetus in the last few years as the result of union activity, so that we are now changing our entire feeling toward employees, that is, the employer-employee relationship is something that has had an entire change-over. In this change-over we have usually transferred this function, where the size of the plant permits, to one individual or to one department, either the personnel director or the industrial relations manager, or, in the small plants, to the actual departmental managers, that is, to the foremen. The best management of personnel in industry is going to develop when the foreman, the actual supervisor who knows the man and who knows the job to be done, is in direct charge of personnel, and that is the objective that most of the more forward looking directors of personnel have.

In this field of management of personnel today we have such basic functions as employment, labor relations, the administration of the safety and health programs and the wage determinations. The subject to be discussed here this morning is the health program in industry. Basically, the personnel director's job is to obtain and maintain a supply of satisfactory workers to operate the various manufacturing units.

Obtaining a satisfactory supply of labor has not been such a great task up until the last year or two. Today, if we can get employees who have two hands and two legs and can hear thunder and see lightning, they are employed; they have a job. By far our greatest endeavor today is the maintenance of a satisfactory labor supply.

We are primarily interested today in a greater output of manufactured goods, that is, productivity is the basic goal. Every employer is interested in how much work an individual is able to do. We have found that in the course of a year industry loses approximately 480,000,000 man days through accidents. Upon closer investigation we find that we lose approximately five times that amount of time on account of sickness. Then, going one point farther, if we could measure the actual days' output lost through inefficiency, we would get a figure that would be immense. However, an adequate scale for measuring this loss has not yet been developed.

We know that the ordinary worker today is producing something less than 50 per cent of what he could produce at his maximum efficiency. Needless to say, that is the problem with which we are faced, and we are constantly trying to improve upon it. We have a tremendous field in which to work, and that field can be approached only by the plant physicians, by those physicians who are associated directly with industry, and by groups such as yours that are interested in industrial employment and the efficiency of workers in industry.

At the time of the pre-employment physical examination, the individual's visual acuity is tested. The biggest task that we have is to get some sort of a vision test which will serve in determining whether an employee's vision is adequate for a certain job. Too often we have used the standard tests as criteria, and to many industrial people 20/20 vision sounds like a football score, but that has been the standard that has been adopted. We may put a man to work where 20/20 vision is no criterion at all when considering the visual performances required for a certain job. Our company makes Spry shortening, and as the product passes by in the lines, inspectors must look closely at it. Visual acuity of 20/20 is not a factor, but we do want somebody who can spot a discoloration in the product and who can spot a black speck coming through. The present visual tests are wholly inadequate for determining the visual acuity necessary to perform some of the jobs that we have.

We supply the plant physician with a full description of each particular job, that is, a description of what actually occurs in each stage of the manufacture of our product, namely, what type of inspection and operation is necessary to perform the work adequately. There are about 180 different types of work. When John Jones comes to the plant physician and says, "I am a soap boiler in the No. 3 house," the physician will thumb through his job descriptions and pick out the description of that of John Jones. The physician's record will indicate whether John Jones has had this particular complaint before. Whatever the complaint is, the plant physician also has a knowledge of the type of work which the employee is doing.

I know that I am getting over into a controversial field, that is, where should the plant physician stop and where should the private physician take over? One could argue about it indefinitely, but of primary concern to us is the fact that each employee is a producer, and anything that we can do to increase his productivity is of interest to us.

If the United States is going to be the arsenal of the Allied Nations, which it is proposed to be at the present time, it is going to mean that there must be maximum production from every employee who enters our mills and factories. That means that we must have close cooperation and coordination between all individuals who are interested in the employee's well being. In other words, everybody who has any contribution to make to the increased efficiency of these employees must work together so that if the personnel man is the final person to confirm the placement of an individual, he should be supplied with information in such form that he can understand, so that he can make this placement to the best advantage of that individual employee and to the company for which he is working. In other words, we do not want to put any employee on a job where he becomes fatigued in 15 or 20 minutes and keep him on that job 8 hours when we know that during 7 hours and 40 minutes of that period his efficiency is greatly reduced.

Official surveys convinced us that that is what is happening today. An inventory should be taken from time to time, that is, compare the employee's productivity with the job requirements and then make the balances. That can be accomplished only through close coordination. If we are going to produce the quantities of materials necessary to supply the Allied Nations, all of us are going to have to hit the ball now and hit it hard. We must get into this thing with a very strong impetus.

We are depending on the employment of women today. We are accepting, also, what generally has been considered the substandard employee at the present time. All of this is part of our effort to increase production. In this type of program it is necessary that the doctor and the industrial people work together, and that they have a mutual understanding of the situations involved so that industrial efficiency can be improved.

CHAIRMAN SNELL: We have the privilege of having another non-medical man on our panel. I take great pleasure

in introducing to you Mr. Cliff Boughner, who is the Safety Engineer of the Carnegie Corporation. He will talk to us on "Eye Safety Programs and Eye Protection from Flying Substances."

MR. CLIFF BOUGHNER: I am interested in one statement that has been made and I would like to discuss it, before presenting the paper that I have prepared. It concerns the statement about the doctor who does not know a great deal about the plant. In my work as safety director I make it my business to see that the doctor knows something about the plant. A plant physician cannot adequately advise what might be the best thing from the consulting standpoint unless he sees the job, so to those of you who are interested in industrial medicine, I would like to suggest that you do see some of the plants.

The other day I got into a part of our plant that I had not seen for 5 years. We have 38,000 employees, and it is quite an undertaking to see what is going on everywhere in the plant at all times. I want to admit at the very beginning that we were the guinea pigs for a test which included nearly 10,000 of our employees, and I believe some of the data presented on this panel pertains to that survey. Although I do not care to name the two participants on the panel who used it, I just feel that some of that material sounds familiar to me. This program was carried out on an around-the-clock basis, and I definitely am of the opinion that it has been a boon to us.

Today, industry, and particularly that part concerned with safety, is more than ever interested in knowing all about its employees. The background of an employee who has figured in a lost-time accident, or in a near lost-time accident, is as much a part and parcel of that report as the fact that the man has had a certain type of injury. The first thing that a good safety director asks for from an employee is a copy of a physician's examination report and a copy of a good eye test, because they can be correlated into evidence that may completely absolve the employee of blame in case of an accident and may definitely place it on management for failure to recognize health hazards.

During the last few years there have been some remarkable advances in medicine, and these have been reflected in better working conditions for the American workingman. Management likes to know what increases their employees' efficiency. If there is a case of eye trouble they like to know about it, because they may be of assistance in improving the employee's vision. Correcting just the visual impairments of employees in a plant, however, will not immediately step up its production 100 per cent. In some plants, regard for the visual performance of employees will result in increased production; in other plants that is not the case; but in general, as a result of this interest in the employee's health the employer-employee relations are improved. These are very important today because of the war effort and because of the fact that industry realizes that it must protect the men who are producing the goods needed to win the war.

I am interested in one other point that was discussed and that concerns the wearing of goggles by workers in the plant. I do not believe that furnishing every employee in a plant with a pair of goggles is the solution to the problem. I do believe that an adequate eye protection program is necessary, however. If a group of employees realizes that the management is personally interested in seeing that they get a good pair of goggles, properly fitted, they immediately become interested in knowing that the corporation for which they work is that much concerned about their personal welfare.

What do I mean by properly fitted goggles? I mean those so fitted that they do not fall off nor are pushed on top of the head at a time when they should be worn over the eyes. Unless the management does its part in seeing that the goggles are properly fitted to employees, many goggles will be found in the men's pockets or in their lockers, and injuries to eyes will occur which when investigated it will be revealed that the goggles did not fit properly. Each pair should be fitted properly to the individual worker. A person who is qualified to fit goggles should be placed in charge of the enterprise. When you want to purchase a piece of meat you do not go to a hardware store. When goggles are necessary for employees, a trained fitter should superintend the project. He is not an eye doctor, but he knows how to fit the cups of the goggles, and he knows how to adjust the bridge and the headband. He should know what type of goggles is required for a certain job, and above all the fitter should have

a personality. It isn't necessary that he wear a white coat so that he will appear to be a doctor, but he should be able to give service. About three days later, the employer should make inquiries, such as, "How do your goggles fit, Bill?" If Bill says, "Oh, they fit pretty good, but the headband seems to bother me," the headband should be adjusted. Already $1.50 has been invested for goggles, but in case of fatal injury to an eye the cost to the employer will be $1,800.

I shall now read a very short paper, which I have prepared.

All management recognizes the need of industrial eye protection. The eye injuries in industry may be divided roughly into five groups:

1. Those resulting from accidents or mechanical injuries due to failure in following safety rules and regulations as set up in the different plants.

2. Those resulting from exposure to injurious radiant energies, such as intense light or heat rays.

3. Those resulting from the use of certain chemicals or other toxic substances.

4. Those resulting from ineffective or improper lighting and other abnormal working conditions.

5. Those resulting from communicable diseases which, while not directly caused by an industrial process, may nevertheless be spread by unsanitary conditions in working places.

Since the beginning of the safety movement, industry has been well aware of the first two of these groups and has concerned itself with protection against them along with other accident prevention. This was so chiefly for two reasons. First, in the first two groups of eye injuries, cause and effect were more readily apparent than they were in the last three groups named, and second, permanent disability occurring as a result of the first two groups of eye injuries was compensable under some of the earliest workmen's compensation laws. Under item 5, industry has recognized the use of preventive sanitation measures, such as goggle sterilization and hygienic measures associated with the general sanitation program.

There is really nothing new to offer in the way of a program of eye protection from flying substances. However, reiteration of those things which have been constantly in mind in the past may, by such reiteration, be regarded as a revitalization of a program. The following suggestions are made:

1. That management continue to recognize that, despite the progress made, there is room for still further achievement in the prevention of eye injuries.

2. That management continue, with zeal and courage, to issue and enforce essential orders and policies to the end that the entire organization — executives, supervisors, foremen and workmen — have before them always as a goal the attainment of complete elimination of accident. This involves continued effort to make each plant, even though it may be already classified in the high brackets of safety, still safer, and that adequate equipment be maintained such as masks, goggles and such other protective material as may be desirable. Today the cost of protective equipment does not constitute a deterrent toward its installation.

3. The keynote in any program is an understanding of safety measures required combined with continuity and periodic inspections of such equipment.

4. As important as the availability of safety equipment is the periodic inspection of such equipment and its maintenance in good order.

5. So as to be on guard against the smug attitude that goes with "It can't happen here," there must be made apparent to supervisors and workers the hazard of eye injuries in industrial occupations and suitable methods of forestalling them.

6. Finally, of course, it goes without saying that unless the workers themselves understand the need for the program, the program itself and, to some extent, the safety equipment will be of little use.

The eyes of the American workingman are more vital to national defense than is the armament which those eyes are helping to build, and the eyes of the world are today on the American workingman. So as I conclude my statement, I merely want to challenge you to find out what is going on in the plants that you service, if you service any of them. Don't sit in your office, but hunt up the safety man and try to find out what the risks really are. You will be a better doctor for the experience, the plant will be a safer place to work in and you will have done your duty to humanity.

CHAIRMAN SNELL: Dr. Kammer, I feel that after all the information that has been given out here, you ought to be prepared to summarize and give us the ideal set-up for a medical organization in a plant.

ADOLPH KAMMER, M.D.: There really isn't much left to say. I suppose it would be a much better handled job if I polished off a few of the good ideas that have been thrown out to you this morning.

All of you who are going to engage in industrial medicine will run into creatures called "medical directors of industrial plants," and in order that you may bear more patiently with some of the obstacles that the medical director will place in your way and in order that you may be more sympathetic with him, perhaps the nature of his job should be presented to you simply as a piece of descriptive material.

The medical director is the health officer of the plant, and he takes care of people who get injured there or who become sick because of employment there. He usually divides his activities into three or four brackets. One of them is either doing or directing a pre-employment examination in order to get employees properly placed. His second bracket is to see that the people who work at the plant are periodically examined with some sort of thoroughness. His third bracket is to see that the people who are injured and become sick there are properly treated, and his fourth bracket, in some ways the most important and in other ways the least important, is to see that claims are properly defended.

You have heard this morning that industry has perforce become more and more lax in the requirements placed on people who are coming up for employment. That is altogether a matter of what the market is, and we now are engaged not so much in selecting people and keeping out those who won't fit anywhere in the plant as we are in guiding them to proper placement and employment.

One of the things that comes into the pre-employment examination is the visual testing, and we did not know very much about how that should be done, and were content with the Snellen wall chart examinations, until the method described this morning got under way. Again it should be stressed that different standards apply to different jobs. In steel manufacturing it seems to me that the standards or requirements for visual performance can be extremely low, and the men can still do good work. That holds true where men deal with things anywhere from 10 to 20 or 50 feet away from them and where all moving objects are large, where lighting generally is pretty fair and where very fine readings are not to be made.

The problem of health maintenance must, of course, embrace visual maintenance, and visual maintenance to me means just part of maintaining a man's health. If I have his eyes examined, I also want his urine examined and his heart examined and his blood pressure taken, and all the things that go to make an adequate appraisal of the man's physical make-up as we now understand it. Now when absenteeism due to illness is a thing which is tremendously expensive, because the ranks of the absent worker can't be filled — there isn't a surplus to take care of that — the general physical make-up of the man needs really to be weighed.

I believe that respiratory diseases are the cause of more absences due to illness than those affecting the visual system. Cardiovascular disability ranks second and perhaps metabolic disturbances and things of that nature, third. So the problem of reducing sickness absenteeism is a problem in which the ophthalmologist can play a part, but it isn't a major part, and the medical director has the job of appraising and orienting the part that each medical specialist plays in the program.

There are many things about workmen and the life of industrial plants that to me are almost not understandable. Professor Tiffin is able to reduce many of them to figures and curves and to point to factors which influence production, safety and things like that. I hope it continues, but at our own plant just now we are finishing up six of the best weeks of safety in the history of the plant. We, the people there, are producing more steel per employee, we have had the fewest accidents in the history of the company and we have the dumbest lot of employees. As to how you can tie that all together and say what is the cause, I do not know. I said to our safety director, "Jake, what has happened to make you so good?" He said, "Doctor, if I knew, we would keep on doing it." When you try to be specific and to say that this or that measure will produce this or that result, you may be wrong or you may be right.

I haven't anything more to say except that I think you will find that medical directors will be willing to accept this program, and that, properly presented and properly applied and properly individualized, it is a program which is going to be useful and is going to make industrial life a little happier.

DISCUSSION

CHAIRMAN SNELL: We hope that the time left will prove profitable. I am sure you have questions that you would like to ask and have answered. You may direct your questions to any member of this panel, personally, or, if you prefer, hand in your questions. Any member of the panel is also free to ask questions.

DR. KUHN: Mr. Lane, do you think it is possible that some day we will be able to write visual skills into job requirements and knit the whole concept thereby much more closely to management's problems?

MR. LANE: Yes, I see no reason why we cannot do that. We are already writing other physical skills into job requirements and job specifications. I think of one instance offhand where we had a packaging operation with packages coming out of the end of the production line at the rate of 80 per minute. We built a packaging arrangement whereby the girls could take these packages as they came down and put them into the cases. We found that a girl 5 feet, 5 inches tall was ideal for that operation, but a good many of the girls whom we employed were 5 feet, 1 inch or 5 feet, 2 inches tall. If they worked on that job they had to reach to put the packages into the case, with the result that there was no inspection. Now we have written into the job specification that the girls for that work are to be approximately 5 feet, 6 inches tall.

I see no reason why we cannot say that a certain type of vision will be required for a job. Our labor contracts today are generally written up with a clause to the effect that the employee taking a job must possess the mental and physical qualifications necessary to handle the work. Now as soon as you have a clause of that kind in your labor agreement or your labor policy, you can go ahead and write your specification, and a visual specification is just as fitting as any other sort of physical specification.

DR. KAMMER: May I ask is your right to include a physical specification ever challenged, and when it is challenged, what kind of an argument do you use to protect your stand?

MR. LANE: Everything that you write into your labor policy today is challenged. You can take for granted when you start writing it that it is going to be challenged.

DR. KAMMER: I mean at the time you reject, we will say, a certain individual.

MR. LANE: We have had a good many cases of rejection that we have had to defend. At the present time, even though that clause I spoke of is 6 years old in our contract, approximately 20 per cent of our workers today are frozen on their jobs. In other words, they are ineligible for promotion because they lack either the physical or the mental requirements to progress. That has meant a good many challenges, but I would say that in 90 per cent of the cases we have been able to defend refusal to let a man go onto a job, either on a mental or a physical basis. In the other 10 per cent where we have been challenged, we have argued it through, and I think that I have had only two where I have had to offer the man a trial and then either accept him or reject him on the basis of the trial.

CHAIRMAN SNELL: There are two questions for Doctor Kuhn to answer. The first is, "How much time can be given to an eye examination?" The second is, "What is the technique for making a near point examination for loopers?"

DR. KUHN: With the exception of a few very special tests, in making the ordinary screening test one can get all the data that one needs in 3 minutes by using our basic battery of tests. For example, more time is required to make tests in near point examinations where an employee is working at 8 inch distances, as the loopers do. Eight inches is not anywhere near a normal working distance, and when we started to check the vision of loopers we did not even know what degree of phoria at 8 inches should be regarded as normal. After determining acuity for near vision at the work distance, phorias for that distance, the near point of accommodation, and the near point of convergence (since there is a

binocular mechanism to be considered), one must then use his judgment whether an occupational lens will be best for the employee. Our data revealed that 67 per cent of the girls, who had normal degrees of phoria at 14 inches, had 20 or 30 degrees of exophoria at 8 inches. For a 20 or 30 degree exophoria at 8 inches one must do something more than magnify and relieve the accommodation. A prism base-in is indicated; but that is a prescription problem in which each case must be studied individually.

It is more difficult for the binocular mechanism to function in very near work distance. A blur is caused by this failure of function, although each eye may be perfect. This point was demonstrated in a plant where a threading operation was so close and so tiny that many employees couldn't see the hole. This was not a question of acuity or of magnification. It was a question of a blur from inability to converge, demonstrated by covering one eye. When one eye was covered, there was no trouble in threading the needle into the hole. These special tests do have to be added to the battery and they take more time.

CHAIRMAN SNELL: Doctor Kuhn, along that line, what has been your experience with the use of prisms, base-in, in cases having a normal muscle balance for distance? In my own experience, I have obtained very satisfactory results by the use of prisms base-in for employees who have to work at this very short distance.

DR. KUHN: At one of these plants that Doctor Tiffin analyzed, where the work was at close distance, a certain number of occupational lenses which incorporated a prism were prescribed with the result that these girls were very much more comfortable. They would leave their glasses on the machines when they were not working as they couldn't use them for any other purpose. They learn by experience that they cannot with comfort look at distant objects.

CHAIRMAN SNELL: Does the three-quarters lens aid in that particular difficulty? With these they can look over the top of the lenses.

DR. KUHN: However, they still have to accommodate.

CHAIRMAN SNELL: I find it rather difficult to get the women to wear the three-quarters occupational lenses, but the men like them, will wear them and they obtain great comfort from them. The women do not like the appearance of these lenses.

There are two questions that deal with radiation that I will ask Dr. Allen to answer.

DR. ALLEN: One of the questions is "Ophthalmia-electrica — possibility of permanent damage." We really need a new revision or a study of the question concerning photo-ophthalmia electrica. A great many statements have been made by people who probably should not have made them, outstanding people like Duke Elder, without adequate scientific evidence to back them up. The article that I mentioned by Verhoeff and Bell, which was written in 1916, is the best scientific investigation to date, in my estimation. A great many others have done some work, but they haven't done it as thoroughly as these two.

It would require about $50,000 to $100,000 and about 3 or 4 years of intensive work on the part of a department of a university to investigate that problem adequately. According to Verhoeff and Bell's article, permanent damage does not result from repeated attacks of electric photo-ophthalmia. The treatment is simply to make the patient comfortable by administering soda biborate and boric acid for a few days, possibly a little castor oil and to have the patient stay at home following which in a few days the situation is well in hand.

The question is also asked regarding the use of vitamins, etc. Of course, Verhoeff and Bell's work was done long before vitamins were known as such, and I don't know that the question has been answered scientifically since then.

When can the patient return to work after the injury? As soon as he is able to open his eyes with comfort.

The other question is, "Have you had cases of ultraviolet burn from the acetylene torch, especially before the torch is used on the metal, that is, while merely adjusting the torch?" I have not seen such cases.

It reminds me of a question that was put to the committee about an alleged injury. As I remember the facts in the

case, they were these: Some workmen were cleaning out the cylinders of Buick automobiles by the use of oxygen and hydrogen, which was ignited by an electric spark producing an explosion. After some months one of the patients had a cataract. Was the cataract the result of the explosion?

This is a situation that we have to consider. It should be the subject of some study by responsible scientific men. In the first place, we have to know the quantity of energy delivered. In the second place, we should know the quality, that is, the exact wave length of ultraviolet or infrared or whatever type of light to which the individual has been exposed. In the third place, we should know the distance of the workman from the explosion and how he is protected, or whether he is protected at all; next, the number of exposures; but most important, it seems to me, is to know exactly the condition of the eye before the workman was put on the job. That cannot be told, for instance, concerning cataract without an examination with a cycloplegic or at least a mydriatic. An ophthalmoscopic examination at that time isn't sufficient either. It should be a slit lamp examination, because some of these very early capsular cataracts can't be seen with the ophthalmoscope.

Other problems enter in, too, as to whether or not the individual might possibly have a hereditary tendency to cataract, or whether he is using some drug at home that in his particular case might cause him to be a little more subject to cataract formation.

I shall give one example: A relative of mine whom I had thoroughly examined 10 years before and in whom I had accurately diagnosed cataractous changes, came in with a fundus lesion that appeared to me to be malignant. I had her examined by a number of other men who agreed with me, but she refused to have the eye removed. She, therefore, had considerable irradiation therapy. The fundus lesion disappeared, and the cataract did not advance, but if we had not known that there had been a cataract 10 years before, we might have thought that the irradiahad produced the cataractous changes.

We know that a good many people in middle life, who have not been exposed to these various irriadiations, have incipient cataracts, particularly of the capsular variety. Therefore, it is incorrect for us as physicians to be very dogmatic about "ophthalmia electra" until we have actually more scientific, reliable information.

CHAIRMAN SNELL: This question has a medico-legal phase in which a causal relationship is difficult to determine. How much and what kind of radiation under given circumstances is considered adequate to produce cataract? For example, I have an employee who is asking compensation, claiming disability because of exposure to electric flashes. The history of the case, briefly, is: He works for a Gas and Electric Corporation, and his job is to repair power lines when they break down. During the last 10 years he has been exposed to repeated flashes. Often his vision has been upset for not more than 24 hours. On one occasion he was hospitalized for about 3 days. The patient has had all the thorough examinations of which Doctor Allen speaks. There is no history of cataracts in the family. The patient has no other complaints. He is 45 years of age, the blood Wassermann is negative and he is developing cataracts in both eyes. In this case, the question is, as an expert, what answer can one give as to causal relation?

We have no definite evidence from an experimental point of view, or from any literature that I have been able to uncover, which says that this kind of exposure to radiation will produce a cataract. Thus, the expert is somewhat on the spot. This may be the procedure to follow or at least it is the one that I follow: The Court is told that a complete physical examination has been made, and an adequate cause for the cataract has not been found. The man has been exposed to heat, light and radiation; and, although the medical profession has not been able to say definitely that such an exposure is the cause of the cataract, it has no other explanation.

Then the usual question is asked, "With reasonable medical certainty, can you state that the accident which has been described produced the cataract?" You can answer that there does not seem to be any very definite medical opinion that is positive, but the repeated exposure may be an adequate or contributory cause for the cataract.

Dr. Kammer has a question.

DR. KAMMER: The question is, "How much does the diet of employees have to do with their efficiency?" I don't know.

There is currently great enthusiasm in this field, chiefly among nutritionists who tell us that we are going to overflow the world with steel if we will just feed our people properly. Doctor Peterson was with a group of authors who made a report in an early issue of the Journal of the American Medical Association this spring. He might summarize that work for us.

DR. CARL PETERSON: Presumably there is a considerable relationship between physical fitness and nutrition. I believe that every physician appreciates that relationship. What we did take issue with was the advice of nutritionists relative to the free distribution, or any kind of distribution, of vitamins for industrial workers. We came to the very definite conclusion that that should only be administered on the basis of definite physical findings.

DR. KAMMER: When prescribing for a deficiency?

DR. PETERSON: Yes.

CHAIRMAN SNELL: Mr. Boughner, you have a question.

MR. BOUGHNER: The question is "From a practical standpoint, how close may unprotected men work to welding operations without danger to their vision?" The question is worded so as to permit me to give a pretty general answer. Unprotected men would evidently mean men not wearing protective eye covering. Shields are used in the welding shop, some of them are stationary and others are movable. They are set up as a protection for all welding operations. A safety line is drawn 20 feet from the point of operation, and a shield must be erected between that space and the actual welding operation. We do not have any unprotected men watching welding operations. I do not think that it is possible to keep men from watching anything that is going on. Open a hood of an automoble down here on State Street and in 10 minutes you will have 50 people watching to see what you are doing. The same thing is true in industry. If the employees see something going on, there is bound to be an interest, but we won't permit radiated light to be reflected so as to harm fellow workmen.

These shields, as I say, are in numerous sections, and when employees go to different jobs in different parts of the mill, it is standard practice to move the sheilds at the same time.

The second part of the question is, "How are goggles sterilized?" Goggles are sterilized in our first-aid hospital. They are placed in a sterilizer. Steam is admitted, and the goggles remain there for a certain length of time. Our first-aid stations are located at our exits and entries, and any man may leave his goggles there on his way out and pick them up on his way in, in the morning, or we may take them to the tool room and the goggles will be sterilized and made available for him in the morning.

CHAIRMAN SNELL: Mr. Boughner, I have a question that I should like to put to you. It seems to me rather important. I am sure that a great many doctors doing industrial ophthalmology have had some difficulty with their goggle program. For example, in one plant, in which we tried to put in a very complete goggle program, all the employees were told, "It is a rule that you must wear your goggles, or else," and we had great difficulty in carrying out that program. Could you tell us briefly how you would manage the employees from a psychological standpoint so that you would get their cooperation and not run into these difficulties?

MR. BOUGHNER: The answer to the question has a twofold aspect. First, you must convince the employee by salesmanship that you are offering him something of great personal benefit to him. The second is a psychologic approach. I ought to relate a personal experience. Some years ago we had quite a bit of trouble around grinders, particularly in our tool rooms, and every once in a while I would go by one of these grinders and find the fellow with his goggles up on his head. Maybe it was necessary that he only touch a tool, to put on a quick edge on it, or maybe he was doing something for which he thought he did not particularly need to have his goggles down. We were having quite a bit of trouble with the program in that particular end of the plant, because we had a young group that had a lot of funny ideas.

All the division safety inspectors throughout our mills carry with them a very small package, which just barely holds a glass eye. When I would go by a machine and find a man grinding without having his goggles on, although he knew

the rule, I would just tap him on the shoulder as I went by him, and when he turned around I wouldn't say anything at all to him, not a word. I would just pass him the little package that has written on it, "An eye for an eye." I was never able to get more than 8 steps away from the man before he would whistle and call me back and say, "O. K., Cliff."

Never say anything to an employee because he has violated a plant rule; since, if you record it on a 426-A form, which is a violation form, and it goes on his personnel record, I think you defeat your purpose to a certain degree by reprimanding him on each occasion. However, if, psychologically, you can bring him to realize that these glass eyes can be purchased for so much a dozen but what he has cannot be purchased for so much a dozen, you have accomplished something.

For a while we used to carry a spark plug with us. When the ordinary layman looks at a gallon of gasoline in a bucket, it does not mean a thing to him, but put a spark to it and it immediately flashes into power. When you furnish goggles you are giving the employee a protection for something that belongs to him, not to you. When you can convince him that you are providing a protective service to make him a better workman, he is going to fall in line with your safety program; but when you can not get cooperation it is best to move such an employee to some place where he cannot fall over the handle of a broom, because eventually he is going to get you into trouble. Employees should not be told, "You must do this."

The thing to do is to convince them that it is for their own betterment. Convince them that you are really doing them a favor by giving glasses to protect them from the possibility of a life of blindness. The whole problem of the economics of that man's life rests on his wearing the glasses which experience has proven to be correct for him to wear. Convince him of this. But don't say, "You wear these, or else." If you do, you are going to find somebody who will go into the "or else" class because Americans do not like to be told that. When you give an employee a pair of goggles, make him believe in them and continue to wear them until he becomes accustomed to them. Don't just walk away from him. Stay with him until you have sold him on the desire to wear them; make him proud of the fact that he is wearing them. If you can once get to the top man in a certain unit, so that wearing goggles becomes more or less the trademark of that particular craft, don't worry about the little fellows, because as soon as they see the big boys wearing them they will wear them, too.

Whenever I make rounds in the plant, I put on my own prescription glasses, and every once in a while somebody will say, "What kind of glasses are those?" I show them to him, and he will say, "Well, those are safety glasses, too, aren't they?"

"I am going to get a pair of those."

In other words, when you mingle with the employees in the mill don't preach something you fail to practice yourself. Don't tell employees that they are forbidden to do something and then violate the rule yourself. Set up a good goggle program and be sure the proper type of goggles are obtained for the particular type of work to be done.

CHAIRMAN SNELL: Dr. Tiffin has a question.

DR. TIFFIN: This question is, "How can visual data on records, where full visual performance or information is obtained, be utilized by the medical profession and management?"

The whole point, as I see it, is to set up a testing procedure, and place it in the hands of someone who is qualified to use that testing procedure, so that he can check the test results against the productive employee and the unproductive employee. It will give to management the requirements, from the visual viewpoint, which supplement the picture of the job that has been obtained from the various other standpoints, physical, mental, etc. In other words, the use of visual data by management is simply to have an objective measurement type of method of setting standards for different jobs instead of standards for jobs based on opinion. It is recognized in every profession that there are cases where there is no objective evidence of what standards there should be or what is the right standard; often one must set standards on the basis of opinion. But everyone will also recognize that very often standards based on opinions are wrong; I mean wrong from the point of view of production. A typical illustration of it is the one mentioned with regard to the

visual requirements of very close workers. It is perhaps not wise to have a standard, based on opinion, of a certain degree of acuity at 20 feet for people doing very close work. It is understandable after the scientific tests have proven the difference, but very few people realized it beforehand, and no one could have realized it ahead of time in terms of the proof that resulted from the standards based on experimental data. The job is to set the standards wherever possible on experimental data rather than in terms of opinion.

CHAIRMAN SNELL: Doctor Kuhn, have you a question?

DR. KUHN: The question is, "Why don't you write myopia, 4 or 5 or 6 diopters, in the job specification for loopers?" Of course, the questioner is assuming that enough myopes are available. I should say that these individuals should not be refused employment because they are affected by myopia, but they should be given an opportunity to do close work for which they are suited.

A second question: "Is it possible properly to care for all types of electric ophthalmia by seeing such patients only once?" I should say not. That depends entirely on the degree of electric ophthalmia present. It is up to the doctor to decide when that patient should be discharged from medical care.

CHAIRMAN SNELL: Dr. Kammer.

DR. KAMMER: I have a question. "Is not the medico-legal responsibility of employers dangerously increased by using in the pre-employment physical examination only a visual acuity test for distance made by non-medical office help?" I haven't had any experience in my claims of that type of examination being used as a defense. It seems to me that there ought not be any more objection to using the record of a trained technician who has made the eye examination than there would be to use the record of a trained technician doing some other kind of work. It seems to me that it is all about the same. The examination is done under the supervision of the doctor who is taking the responsibility for it and who is testifying.

DR. R. O. GRIGSBY (Ashland, Wis.): The findings of a school nurse will be accepted in Wisconsin.

CHAIRMAN SNELL: May I answer that question in part? I think it depends upon the Courts in the various states. In a good many Compensation Courts only the testimony of the doctor is considered adequate, as it is considered the best testimony. However, in most of the Civil Courts, any person who qualifies as an expert may give testimony and the Court will say, "We will accept that for its value, whatever the value may be."

MR. BOUGHNER: What makes an expert?

CHAIRMAN SNELL: Qualifications are brought out by the questioning of the Court or of the attorney. Supposing you were a real estate man and you put a value on a piece of property. You would be asked, "What has been your experience? How many years have you worked in this particular field and what are your qualifications?" One generally must state his qualifications before qualifying as an expert in any field.

Here is another question. It has been suggested that I answer it. The question deals with the treatment of foreign bodies in the cornea. "In a paper, written some time ago, it is stated, 'when there is a burn of the cornea, if it is treated with full-strength carbolic acid, the burned area will heal quickly and efficiently'."

I would answer this question thus: Any injury to the cornea which is over the pupillary space should be treated very conservatively. By that I mean, you do not want to make a greater injury and more traumatism by using carbolic acid or anything else. I think that these foreign bodies and burns should be removed under magnification as carefully and as completely as possible without destruction of any normal tissue. A great many of these burns are sterile. If you cannot remove all the little brown rings, that sometimes remain, just let them alone for two or three days and nature will do the job. About the second or the third day after the accident, that little ring will come out very easily. In others words, avoid adding to the traumatism by unnecessary treatment.

Some of the detail men have for sale little burrs to be used in removing burns in the cornea. I regard this as a most damaging instrument. However, if the burn is outside the corneal space, one may use his judgment, and, if one can see the patient only once and must do a really

clean job, perhaps one is justified in using the more radical methods. One might even be justified in putting a conjunctival flap over the injury. I have seen that done, although I think it rather extreme surgery.

Another question has been asked. "In these cases of trivial foreign bodies in the cornea, should the eye be tied up?" There seems to be quite a dispute as to the proper practice in such cases. I find that when employees go to first-aid hospitals, even if they have only a very trivial superficial abrasion in the cornea, they always come away with a patch over the eye and with a dilated pupil, showing that atropine has been used. Atropine is not indicated in the first-aid treatment of a superficial foreign body in the eye. I do not find it necessary to bandage an eye in an ordinary case of foreign body in the cornea when the injured person returns to work immediately. If the injury as a result of the foreign body is sufficient in depth and extent so that you think the injured person is not able to return to his job, then it may be good practice to apply a bandage. In others words, you must use your medical judgment in each case and not follow a definite rule.

Doctor Hitz has a question.

DR. HITZ: "What provision is made in the Army for case-hardened lenses?" I have not been in the Army long enough to be certain that I can answer that question. We prescribe government issued lenses and we prescribe colored lenses. As far as I know we are able to obtain hardened lenses for mechanics in the air corps.

DR. ALLEN: How about the fields in the three minute test?

DR. KUHN: In certain cases it is important that the fields be tested, and in old people I certainly do agree with Doctor Allen that more attention should be given to the fields. A simple confrontation test is an ample screening test which the technician must be trained to do, but I do think that fields are important and that final testing and interpretation must be done by ophthalmologists.

DR. F. N. KNAPP (Duluth, Minn.): How about intra-ocular tension?

DR. KUHN: Actually, these aren't medical examinations. Theoretically, a complete examination should be made of every person who is accepted for employment in an industry, if it is to be stated that that person has normal eyes, but this is not being done in making these vision tests. We are trying to sift out everybody who has obvious trouble and to correct it. It is important to determine the intra-ocular tension, but this cannot be done by laymen.

DR. HITZ: To me of utmost importance in any kind of a battery of screening tests is to impress the individual that his passing the visual test does not indicate that his vision is perfect. None of those tests should be so interpreted. But unless that is impressed upon the examinees, incipient glaucoma, or a great many other diseases may be neglected, because the technician in the shop said their eyes were all right.

In Milwaukee, we tried to impress upon the employees who had the test that this was done to help them in their work. If they did not pass, they were requested to go to an oculist, but if they did pass, they were informed that it did not mean that their eyes were perfect.

DR. KUHN: Our plan in this testing program is for the technician not to say anything to anybody, but that the interpretation of the report be made by the medical department, or at least guided by the medical department. The data will indicate that an employee has passed the visual requirements for the job. That is about all there is to it.

DR. P. G. MOORE (Cleveland): Dr. Snell, we express our appreciation of your efforts in getting this panel together and bringing these experts here. We certainly are indebted to you and to them.

Society Proceedings

CANADIAN OPHTHALMOLOGICAL SOCIETY

Fifth Annual Meeting
Montreal, September 21, 1942

Scientific Proceedings

The clinical meeting was held at the Royal Victoria Hospital, in the Out-Patient Department. Many interesting and instructive cases from the city hospitals were presented by members. Demonstrations and discussions were informal.

Scientific Session

Montreal Neurological Institute

The first paper was presented by Dr. William Cone, of Montreal, the guest of honor, who spoke on "The Present Status of the Local and Systemic Use of Sulfonamides."

Sulfanilamide was first synthesized in 1908, and it was refound in 1931. Experiments in 1932 were of decisive importance. Prontosil is a combination of azodyes and sulfanilamide. The azodyes are merely secreted. They have been found to be of no use at all.

In England, Paul Fildes, working on dietetics of bacteria, found certain things necessary for bacterial growth. (Paul Fildes: Lancet, 238:955-957, 1940.)

These are an "essential metabolite" and a "growth factor." The former is a substance or chemical group which takes an essential part in a chain of synthesis necessary for bacterial growth. The growth factor is an essential metabolite which the cell cannot synthesize. Nicotinic acid, for instance, is an essential metabolite for all bacteria, but a growth factor for only a few. Antibacterial substances function by interfering with an essential metabolite and thus inhibit growth. P-aminobenzoic acid was found to antagonize sulfanilamide. This P-aminobenzoic acid is supposed to be an essential metabolite. We should know how other drugs act on sulfanilamide — such as quinine. An approach can be made by this study of dietetics of bacterial growth. The metabolism of bacteria is similar to cell growth, but normal cells have more reserve, that is, the inhibitory effects of cell growth are less drastic than of bacteria. The measure of the effects of all the sulfonamides can now be done, based on this work of Fildes.

In the local use of sulfanilamide in wounds, the crystals were found to be of various sizes. The finest micro-crystals were found to be best for wounds. For infected head wounds, wet dressings of sulfanilamide, cod liver oil and honey have given best results.

Cases of head injuries with infected wounds treated by such packs were illustrated. The group showed that with the local use of sulfanilamide in this way, the wounds healed very well in a surprisingly short time.

DISCUSSION

Dr. Nicholls asked for details of the preparation of the gauze.

Dr. MacMillan asked how long the gauze pack was left in the wound and if it became adherent.

Dr. Cone: The gauze is made with alphamel paste and micro-crystals of sulfanilamide. All three substances are sterile. Honey is hydroscopic and makes a better emulsion than oil alone. It prevents adherence and is removed after 10 days.

"Unusual Associated Changes in a Case of Krukenberg's Spindles," Dr. W. Gordon M. Byers, Montreal.

A white female, 43 years of age, had first noticed blurring of vision of the left eye two weeks before examination.

Examination revealed an almost vertical Krukenberg spindle, 4 × 1½ mms., in the pupillary area of each cornea. To the naked eye, the spindles seemed to be smooth, but with the slit-lamp (Oc. 2, Obj. a2) they resolved themselves into a multitude of minute, rounded, golden-brown pigment dots, which, most densely deposited on Descemet's membrane behind the spindles, became more widely distributed toward the limbus, and seemingly disappeared about two-thirds of the way in that direction.

A second finding, associated with the Krukenberg spindles, was a ring of opacification about the whole periphery of each

cornea, just inside the limbus. The rings were not solid lines, but loosely composed of dull, dusty particles that resembled commas, rosettes and the like. Their edges and their surfaces seemed wavy, and gave the general appearance of a wreath.

One could explain the position and the pattern of the rings only by the presence of the pigment deposits in the meshes of the pectinate ligaments, and it was evident that retro-illumination and an open anterior chamber were essential to their complete visibility. Direct examination of the area of the ligamentum pectinatum will, however, show the deposits in part even in the presence of adhesions between the iris and ligament. In a recent study, Evans, Odom and Wenaas mention that in 202 cases of Krukenberg's spindles there was a pigment band of this nature in 3 cases.

The third feature of the case was secondary glaucoma. The right fundus was normal; left fundus: moderate cupping of the optic disc. Central vision: O.D. 45 + 0.37, 6/6—; O. S. 70 —0.37, 6/9 full. Fields of vision: right eye, completely normal; left eye: general constriction, with some splashed scotomata that were due in part to obstruction by pigment deposits. Tension: right eye, 38 Hg. mm.,; left eye, 45 Hg. mm.

A corneoscleral trephining was done in this case on both eyes, with no complications, and with satisfying results to date.

DISCUSSION

Dr. MacMillan asked if Krukenberg's spindles associated with glaucoma had been noted previously.

Dr. MacDonald thought the difference in color of the pigment rings from the spindles indicated that it was of longer standing or due to currents in the anterior chamber.

Dr. Byers stated that secondary glaucoma was noted in 15 per cent of the cases.

"Exploration of the Orbit and Removal of Foreign Bodies and Tumors by External Canthotomy," Dr. J. A. MacMillan, Montreal.

The simplest approach to the orbit is of the greatest importance at all times in ophthalmic practice, and it is particularly so in war time when an increase in the number of cases manifesting foreign bodies in the orbit is to be expected.

The approach to the orbit through the conjunctiva, after an external canthotomy, as described by Lagrange (Cong. Franc. de Chir. proc. verb., 6, p. 189, 1892) is highly recommended for removal of foreign bodies and tumors as well as the simplest method for exploration of the orbital cavity. The author agreed with Reese (Trans. Am. Acad. Ophth. and Otolaryng. p. 76, 1934) that a tumor can be removed as satisfactorily in this manner as by the Kronlein method, which is a more extensive procedure. He disagrees with Stallard (The Surgery of Modern Warfare, edited by Hamilton Bailey: Livingstone, Edinburgh, p. 599, 1941) who says that when foreign bodies are situated 2 cm. or more behind the orbital margin the retro-orbital contents must be explored by means of a Kronlein operation. In proptosis due to diffuse hemangioma of the orbit, the use of radon seeds is advised. Cases were reported.

DISCUSSION

Dr. Morgan agreed with Dr. MacMillan that the simplest operative method was the best, and he quoted Dr. Dunnington to the effect that a Kronlein operation makes a difficult operation just that much more difficult.

"Self-Inflicted Ocular Mutilation, Resulting in Enucleation," Dr. A. E. MacDonald, Toronto.

Three enucleations were required for two males, 32 and 33 years of age, after they had mutilated their visual structures. The remaining eye of one patient had a severe hemorrhage into the anterior chamber, which reduced his acuity to the ability to count fingers at two metres. With hospital treatment, normal vision was restored for this patient, who has since left the country.

The other patient had injured the four lid margins as well as the two globes, in 1926. Since that time he has been employed as a basket weaver with the Canadian National Institute for the Blind, where his conduct and work have been satisfactory. The three globes that were removed had been ruptured in situ, accompanied by uveal prolapse, principally by the use of the thumbs.

"Depth Perception in Aviation," Squad. Leader John V. V. Nicholls, No. 5 Medical Selection Board, Belleville.

A theoretical and practical discussion was presented of the factors in depth perception as they affect aviation. These factors include both monocular and binocular acuity. The two are integrated by a complex psychological process, which is affected by many phenomena. Experienced pilots depend mainly upon monocular clues. Binocular factors have their greatest importance during the early stages of flying training. Development of monocular acuity can compensate for the loss of binocular vision. The delicacy of binocular depth perception, or stereopsis, has no relationship to the amount of heterophoria present per se. The stability of the heterophoria present and the dependence of the pilot upon his binocular clues are the important matters.

Dr. MacDonald discussed kinetic stereoscopy in motorists, but this does not apply in the air where there is no comparison of fence posts, trees, etc. A man with a squint and central suppression still has better flying ability than a one-eyed man because of the preservation of the field of vision.

"Malignant Exophthalmos," Dr. J. F. A. Johnston, Toronto.

Six cases of malignant exophthalmos were reported which were treated by decompression of the orbit.

In one case, vision was lost by perforation of the cornea before the patient came under observation.

The results in all cases showed a marked reduction in the exophthalmos with preservation of vision. Following the removal of the supra-orbital plate, in one case, a complication developed, which consisted of a large blood clot over the right frontal lobe.

In reviewing this group of cases as a whole it is noted that epiphora is present long before exophthalmos occurs. Exophthalmos may appear a long time after a thyroidectomy. Decompression of the orbit is not complicated by shock or suffering. The eye is restored to the orbit, and normal vision is maintained.

"Handedness and Dominant Eye," Surg. Lt. J. M. Parker, R.C.N.V.R. From the Royal Canadian Naval Medical Research Unit. Banting and Best Department of Medical Research.

A report was presented concerning the examination for ocular dominance, apart from handedness, of 545 men with 6/12 vision or better in each eye, who were free from squint. Mid-line readings were not noted when the curiopter was used, described by MacDonald (Trans. Am. Ophth. Soc. 39:214, 1941), and the subject's opinion as to handedness was accepted.

The results showed high ocular dominance for 75 per cent. Of these, 70 per cent were right-handed, and 5.2 per cent left-handed. Left ocular dominance was found in 25 per cent. Of these, 20.2 per cent were right-handed, and 4.6 per cent left-handed. In only 1.1 per cent was there a history of changed handedness.

"Significance of Aniseikonia in Aviation," Flt. Lieut. A. J. Elliott, Medical Branch, Royal Canadian Air Force, Montreal.

The importance of aniseikonia in the judgment of distances while flying has been a matter for conjecture among investigators in the field of aviation medicine for the past seven years. For this reason a group of 175 ex-training pilots, in whom the judgment of height and distance was incorrect while landing, was examined for the presence of aniseikonia. A control series of 50 experienced pilots was similarly studied.

The examination was performed with the space eikonometer, developed by the Dartmouth Eye Institute. The aniseikonia was measured in the vertical and horizontal meridians, and cyclophoria determinations also were made. The amounts of aniseikonia were found to be exceedingly small in both the ex-training pilots and the control group of experienced pilots.

A series of landings was made in aircraft in order to study the practical effect of various aniseikonic lenses on the judgment of height. The importance of monocular visual clues was noted.

It was concluded that no significant correlation existed between the presence of aniseikonia and the landing performance in the two groups studied.

Section on Ophthalmology of the American Medical Association

SCIENTIFIC EXHIBITS

ATLANTIC CITY, NEW JERSEY
June 8-12, 1942

NEURO-OPHTHALMOLOGY

DONALD J. LYLE, M.D.

In this exhibit an attempt was made to correlate clinical ophthalmology with the neurological and roentgenological examinations and to establish the clinical diagnosis by the findings of the neurosurgeon and pathologist.

A section was devoted to the common syndromes of the brain and brain stem pertaining to ophthalmology. The charts showed the anatomical location and the physiological action and presented a pathological case to demonstrate the clinical importance of the syndrome.

Anatomical brain dissections and pathological brain sections were demonstrated. Composite drawings were presented of dissections and specimens showing the visual system, the blood supply of the brain and eye, and the cerebrospinal circulatory system in reference to ophthalmology.

PREMATURITY AND EYE ANOMALY
Retrolental Fibroplasia

T. L. TERRY, M.D.

The exhibit illustrated multiple, bilateral eye anomalies in seven premature infants. The appearance of the anomalies at various stages including partial involution was shown. These developmental aberrations, first noticed when the infants were from four to six months of age, were:

- retrolental fibroplasia (opaque mass of embryonic connective tissue behind the crystalline lens)
- shallow anterior chamber
- persistent hyaloid artery
- congenital retinal folds
- adduction of optic nerve head to lens (through traction of unyielding remnants of hyaloid arterial system)

Illustrations were included of similar conditions in infants born at full term. These pictures were from sections of eyes usually enucleated because of a clinical diagnosis of retinoblastoma. In these cases the condition was discovered shortly after birth and was limited to one eye. The illustrations showed a mass of embryonic connective tissue behind the lens resulting in distortion of ciliary processes, congenital cataract formation and microphthalmos. One photograph of tissue removed from an eye with "congenital cataract" showed a mass of embryonic connective tissue attached to portions of ciliary processes and retina.

Since this disease entity is related to persistence of the hyaloid artery and tunica vasculosa lentis or the abnormal involution of these vessels, stages in the normal development and regression of this system were illustrated. Foetal pig eyes, the vessels of which had been injected with India ink, were demonstrated under the dissecting microscope. Various stages were illustrated by drawings and photographs, one drawing showing the close correlation between the vessels of the intermediate portion of the tunica vasculosa lentis and the valleys of the ciliary processes. This system in humans, pigs and cats was shown in three dimensional form by reconstructions. These were made by projecting sections onto a ground glass plate and tracing the vessels on sheets of cellulose acetate. The sheets were carefully aligned and the proper depth was obtained by placing mats between the sheets. Sections used in making these reconstructions were available for microscopic study.

MACROSCOPIC AND MICROSCOPIC OCULAR PATHOLOGY

G. A. SHETTER, M.D.
BERNARD SAMUELS, M.D.
CONRAD BERENS, M.D.
BRITTAIN PAYNE, M.D.
and
E. B. BURCHELL, M.D.
New York Eye and Ear Infirmary

Exhibit showing gross pathology consisting of mounted hemisections of globes, photomicrographs of unusual slides and differential staining.

INTRANASAL DRAINAGE FOR CURE OF CHRONIC TEAR SAC INFECTION

DAVID J. MORGENSTERN, M.D.

The exhibit consisted of diagrammatic illustrations of stages of procedure; instruments and demonstration on skull showing suitability of this technic, which is accurately guided by finger palpation

at the inner canthus for difficult cases as in (1) narrow or fractured noses, (2) small thick-walled, granulation-filled adhesion-bound sacs, (3) acutely inflamed sacs with threatening complications securing immediate drainage with the least possible surgical intervention and almost simultaneous sealing of vascular channels; adaptability in reestablishing intranasal drainage in previously excised tear sacs; summary of the advantages of this technic.

CHORIORETINAL ARTERIOLAR NECROSIS IN MALIGNANT HYPERTENSION
REPORT OF A CASE

MARTIN COHEN, M.D.

Exhibit of colored drawings of the fundus and reproductions from photomicrographs of the retina, choroid and kidney representing the severe form of malignant hypertension. The observations in this case demonstrate the fact that arteriolar necrosis of the retina and choroid may occur in the absence of similar vascular changes in the kidney or in any other organ.

In malignant hypertension the fundus diagnosis may be: malignant hypertensive retinopathy or malignant hypertensive neuroretinopathy, both occurring in the moderate form, or malignant hypertensive retinopathy with papilloedema which occurs in the severe form. (Published in the Archives of Ophthalmology, May, 1940, 23:1052-1059.)

The Fundus Oculi in Hypertensive Vascular Disease

MARTIN COHEN, M.D.

Exhibit of 3 colored fundus drawings from a case of hypertensive disease observed in the benign and malignant phase with abstract of the case. The fundus changes were observed over a period of five months. The fundus classification which I have adopted, based on clinical and pathological studies, is as follows: In benign hypertension the fundus diagnosis may be benign hypertensive arteriolar sclerosis or a benign hypertensive retinopathy. In malignant hypertension the fundus diagnosis may be malignant hypertensive neuroretinopathy which occurs in the moderate form, or malignant hypertensive retinopathy with papilloedema which occurs in the severe form. It is difficult to define the transition of fundus lesions from one form into another; progression of these lesions will determine the classification. (Dr. Cohen's article on this subject has been accepted for publication in the Archives of Ophthalmology and will appear in the near future.)

THE ZONULAR CHAMBER OF THE EYE

HENRY MINSKY, M.D.

Exhibit showing the lens-vitreous system isolated from fresh eyes by removing in saline solution the overlying structures. The vitreous hangs from the suspended lens by means of the zonular membrane's insertion into the vitreous at the hyaloretinal line. Pathologic slides are shown as presumptive evidence of a space being present in life. Final proof is offered of the existence of the zonular chamber by showing its being injected with carmine in fresh human eyes. From one single point of injection a posterior sinus beyond the processes fills up. This communicates by linear channels in the valleys of the ciliary body with the circumlental space. The anterior chamber fills at the same moment, showing that there exists a free opening of the zonular chamber into the posterior chamber. The role of the zonular membrane in accommodation, cataract extraction, detachment of the retina and glaucoma is suggested.

SULFONAMIDE THERAPY IN CLINICAL AND EXPERIMENTAL OCULAR INFECTIONS

PHILLIPS THYGESON, M.D.

The experimental part of the exhibit was subdivided into:

1. Experiments in vitro demonstrating the bacteriostatic effect of sulfa drugs on the bacteria most frequently found in ocular infections.
2. Experiments in vivo on the effect of inflammation upon the absorption of sulfanilamide in rabbit's aqueous using sulfanilamide powder in the conjunctiva. It was concluded that local chemical and bacterial irritants accelerate absorption (damage to corneal epithelium). Protein shock retards it.

3. A study of the applicability of the iontophoretic introduction of the soduim salts of sulfathiazole, sulfapyridine, sulfadiazine, and sulfacetamide. It was demonstrated that with this method much greater concentrations of the drugs were achieved in the anterior segment of the eye than with other clinical methods. Concentrations are maintained for about four hours after a single application.
4. Kodaslides illustrated the effectiveness of iontophoresis with sodium sulfadiazine alone and combined with the oral administration of sulfadiazine on the experimental Pyocyaneus keratitis of rabbits. The superiority of the combined treatment over some other forms of sulfonamide therapy in this infection was demonstrated.

The clinical part of the exhibit consisted of a report of the results obtained in oral sulfonamide treatment of 153 ocular infections, illustrated in part by projected kodaslides. Good results were obtained in a high incidence of infections of the lid border, tear sac, conjunctiva, cornea, and in endophthalmitis due to Staphylococcus aureus. In the tabulation the etiology of the disease was specified. Indications for local or general or combined local and oral therapy were outlined.

EXPERIMENTAL INTRACAPSULAR CATARACT EXTRACTION SUSPENSORY LIGAMENT AND HANOVER'S CANAL

JACOB GOLDSMITH, M.D.

Exhibit dealing with further studies into the dynamics of the intracapsular cataract extraction, such as tumbling of the lens, external application of hook inferiorly, separation of lens from vitreous (zonular fibers severed previously) and so forth. Further investigations into Hanover's canal, such as contents, structure of its walls and so on. Serial microscopic sections from a normal adult eye and also from a normal adult eye which had undergone an intracapsular extraction (sagittal, transverse and coronal cuts). These sections take up the complete zonule of Zinn and all its closely related structures (lens, vitreous, ciliary body).

CRICOPHARYNGEAL SPASM

LOUIS H. CLERF, M.D.
and
F. J. PUTNEY, M.D.
BY INVITATION
PHILADELPHIA

THE TERM cricopharyngeal spasm often is used synonymously with functional dysphagia, spasmodic stenosis, globus hystericus, hysterical dysphagia and esophageal neurosis. It commonly is described as a "lump in the throat" and as a symptom of disease of the upper end of the esophagus. The difficulty in differentiating between functional disturbances and organic diseases at the level of the cricopharyngeus muscle probably has contributed to this apparent state of confusion. In addition, the lack of interest manifested by physicians in diseases of the upper end of the esophagus and their tendency to ascribe symptoms of an indefinite character to functional or emotional disturbances has been an added factor. It would seem desirable therefore to review this subject and study the behavior of the cricopharyngeus in health and in disease.

The cricopharyngeus muscle consists of the lowermost or transverse fibers of the inferior constrictor muscle of the pharynx. It is attached laterally to the cricoid cartilage and is inserted posteriorly in the median raphé of the pharynx. It can be considered as a sling-like muscle which surrounds the posterior and lateral aspects of the cricoid cartilage and the upper orifice of the esophagus. It does not encircle the esophagus. Above, it is continuous with the oblique fibers of the inferior constrictor of which it is a part; below, it merges with the circular muscle fibers of the esophagus. The circular muscle consists of striated or voluntary fibers and has an innervation similar to that of the cricopharyngeus. The inferior constrictor muscle including its transverse fibers as well as the circular muscle of the upper esophagus are innervated through the pharyngeal plexus which receives nerve fibers from the glossopharyngeal, vagus and sympathetic nerves.

While the cricopharyngeus, which commonly is considered as the sphincter of the upper end of the esophagus, plays an important part in the act of swallowing, it apparently makes no contribution towards propelling food from the pharynx into the stomach. This function is accomplished by the action of the three constrictor muscles of the pharynx as well as the muscles of the esophagus. At the approach of a bolus of food the cricopharyngeus relaxes and allows it to enter the esophagus. According to Negus,[9] an important function of this muscle is to keep the esophagus tonically closed to prevent air from being inspired into the esophagus where it would serve no useful purpose and further would interfere with normal respiration, in that it would cut down the tidal volume of respired air. It is very probable that the circular muscles of the esophagus subjacent to the cricopharyngeus also serve a similar function for in laryngectomized patients in whom the cricopharyngeus has been severed from its attachments to the cricoid cartilage there normally is little aerophagia or distention of the esophagus.

Presented at the Forty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 11-14, 1942, Chicago, Illinois.

While the action of the cricopharyngeus can be studied by direct endoscopic and roentgen ray examinations, slight disturbances usually cannot be observed, as the process of swallowing is rapid in this location. Marked narrowing of the lumen, the result of physical changes, can be readily detected in the lateral and in the postero-anterior position, as the stream of barium will be seen to be narrowed. To ascertain the presence of slight changes Mosher[8] has emphasized the importance of centering the fluoroscopic screen or the film on the larynx.

While esophagoscopic examination commonly is employed to ascertain the presence of disease or spasm of the esophagus, considerable experience is required to distinguish between the normal and the presence of spasm in an unanesthetized patient. Usually the sphincter remains tightly contracted and will relax only on swallowing or on deep inspiration. General anesthesia or profound narcosis will relax the normal cricopharyngeus and thus assist in differentiating between the normal and the spasmodically contracted or organically narrowed cricopharyngeus.

Although Elze[2] believes that the true functional constriction producing the characteristic stellate lumen as seen esophagoscopically is formed by the upper circular fibers of the esophagus, a majority of esophagoscopists consider the upper sphincter of the esophagus as the cricopharyngeal fold which is produced by the cricopharyngeus and is posterior to the lumen of the esophageal orifice. It is for this reason that insistence is made on keeping the lip of the esophagoscope anteriorly when passing through the cricopharyngeal constriction. It must be admitted, however, that the upper circular fibers do exert a constricting effect since the lumen of the cervical esophagus does not balloon up as readily as the thoracic esophagus when the cricopharyngeal fold is passed.

Since the normal function of the cricopharyngeus is to maintain closure of the upper end of the esophagus and to relax only to permit passage of fluids and food, any abnormal contraction or spasm will interfere with its relaxation during the act of swallowing, or may give rise to painful sensations referred to the region of the upper end of the esophagus. It is apparent that any act as complex as swallowing would be influenced by a wide variety of stimuli to any one of the many central or peripheral nerve pathways. Since the circular muscle of the upper portion of the esophagus is made up of striated fibers which are closely related to the cricopharyngeus and have practically a common nerve supply it should be necessary to consider this in discussing spasm. It also would appear necessary to ascertain whether there is excessive muscular contraction present constituting true spasm or whether the disturbances are due to failure of a tonically closed normal sphincter to open. There is no known method of examination which will afford this information. Beatty and Palmer[1] have referred to certain patients who cannot swallow tablets or capsules and ascribed their difficulty to faulty management of the tongue or oral muscles in the act of deglutition. As soon as the object reaches the pharynx it is swallowed without difficulty. Small quantities of barium often are retained in the hypopharynx for brief periods during a functional study of the esophagus. This cannot be interpreted as spasm since a considerable portion of the mixture already has passed into the esophagus. Temporary lodgment of a barium-filled capsule in the esophagus often is construed as spasm. This is especially common in the roentgen ray study made following removal of an esophageal foreign body. There usually is present inflammation and edema which can interfere with the passage of a cap-

sule and also may initiate spasmodic contraction of the circular muscle fibers.

It is difficult to accept cricopharyngeal spasm as the cause of lodgment of foreign bodies in the cervical esophagus. A majority of these objects are below the cricopharyngeus and are held by the circular muscle fibers of the esophagus or more probably by a combination of muscular contraction and a crowding of the esophagus at the upper narrow aperture of the thorax by periesophageal viscera. It is a common experience to have the foreign body descend rapidly when once dislodged from the cervical esophagus.

Nonstriated muscle fibers which form the walls of practically all the contractile tubings and sacs of the body and a majority of the sphincters which guard these are frequently the seat of spasm. While this form of misbehavior is not so common in striated voluntary muscle fibers, it can and does occur as is evidenced by the larynx when foreign material is aspirated into the airway. Nonstriated muscle fibers are definitely susceptible to psychic factors. Faulkner and his associates[3,4] have demonstrated by roentgen ray examination as well as by esophagoscopy that disturbing emotional influences appear to be underlying factors in the production of esophageal relaxation or spasm. They were able at will to produce esophageal spasm by inducing thoughts of insecurity and frustation while suggestions of a pleasant character caused esophageal relaxation.

In a review of the literature on disturbances of the cricopharyngeus muscle one is impressed with the many conflicting reports. In the syndrome designated as Plummer-Vinson's[7] the cricopharyngeal narrowing has been described variously as being due to functional stenosis, webs, or changes in the mucosa and submucosal layers. Vinson[12] considered the disturbances as functional. Mosher[8] stated that in a majority of the patients he examined there was a physical basis for the symptoms, namely a web, and that treatment of this gave prompt relief. While Kelly[6] and Paterson[10] believed that the disturbances may be due to spasm, Paterson observed that a loss of the normal sphincter-like form of the mouth of the esophagus is not unusual, the opening appearing as a pin hole, a slit or an irregular opening. There is a feeling of resistance when the esophagoscope is passed which probably is due to a lack of resiliency, the result of thickening of the submucosa, as was observed by him in the lingual and buccal mucosa. Rodgers[11] believed that the innervation of the sphincter is under control of the sympathetic component of the pharyngeal plexus derived entirely from the superior cervical ganglion. He successfully treated such a case by sympathetic denervation and reported an excellent functional result. We are of the opinion that there is definite organic disease at the cricopharyngeal level in these cases and while there may be an element of spasm present, this does not account for the dysphagia nor can relief by esophagoscopy be explained on this basis alone. This difference of opinion by a number of observers suggests that visual examination of the upper orifice of the esophagus is not entirely satisfactory and that it would be extremely difficult to detect by this means alone failure of a normal appearing cricopharyngeus to relax.

One often is tempted to make an inferential diagnosis in patients who complain of indefinite, indescribable sensations about the throat which may be associated with the swallowing of food and often are more distressing when saliva alone is swallowed. Many of these conditions already have been diagnosed as hysteria and the question immediately arises whether it would be desirable to carry out further investigations including esophagoscopy and by so doing "fix the neurosis," or to ascertain if the

functional disorder is due to organic disease. The following case is cited:

Case No. 1. A woman, aged 48 years, employed for many years as a school teacher, complained of a lump in her throat which could not be localized accurately, did not interfere with swallowing of food, was equally distressing when saliva was swallowed and was aggravated by emotional upsets and physical fatigue. She exhibited evidences of a neurotic personality and presented certain of the so-called "stigmata" of hysteria. This diagnosis appeared to be amply justified. A roentgen study of the swallowing function was not satisfactory due to her failure to cooperate. The desirability of esophagoscopy was debated, but finally was performed and an early postcricoidal carcinoma was found.

Comment: The growth was not obstructing sufficiently to interfere with the passage of food. Its presence apparently excited reflex contractions of the striated circular muscle fibers of the cervical esophagus which are supplied by the same nervous mechanism as is the cricopharyngeus and these accounted for the subjective disturbances in the swallowing function.

It is very probable that these reflex effects may be referred from more distant points. Instances of symptoms referable to the upper esophagus in diseases of the stomach, duodenum or biliary apparatus have been cited. The following case is illustrative:

Case No. 2: A man, aged 54 years, who recently had incurred serious financial losses, complained of attacks of difficulty in swallowing which were referred to the upper esophagus. These attacks came on without warning, were of brief duration, unassociated with pain and suggested complete obstruction of the esophagus since the patient was unable to swallow even his saliva during these episodes. A roentgen ray study of the swallowing function revealed a normal cervical and upper thoracic esophagus. Some change was observed in the lower third of the esophagus which suggested ulceration, as a small quantity of barium adhered to the esophageal wall. The patient failed to respond to antispasmodics and general medical care and finally esophagoscopy was recommended. Examination of the esophagus revealed an early carcinoma involving the anterior wall at the junction of the middle and lower thirds. This was nonobstructing and apparently did not interfere with the passage of food.

Comment: It was believed that irritation of the carcinoma was responsible for the reflex disturbance in the upper esophagus which could be interpreted as failure of the cricopharyngeus to relax during the swallowing act and possibly contraction of the circular esophageal fibers. Whether it was due to increased contraction of these muscles or there was a dissociation of the swallowing mechanism could not be determined. There was nothing to suggest a diagnosis of hysteria.

A similar mechanism has been suggested as responsible for the development of pulsion diverticulum of the pharynx. Incoordination of the swallowing mechanism either by failure of the cricopharyngeus to relax or increased tonic contraction of its fibers would temporarily prevent the progress of a bolus of food which is being propelled onward by the forceful contractions of the pharyngeal constrictors. This could be compensated for only by a bulging or stretching of the pharyngeal wall particularly at the weak point in the inferior constrictor muscle between its circular and oblique fibers posteriorly. Patients occasionally describe a momentary "cramp" of the throat muscles especially when swallowing fluids and often have a severe choking attack due to aspiration of fluids. It is

conceivable that this would occur in cases of chronic hypopharyngitis.

We have observed several cases of disturbed swallowing unassociated with obstruction which could not be explained by roentgen ray examination and esophagoscopy. All were middle aged men who exhibited certain neurotic manifestations which could be diagnosed as functional dysphagia. Systemic studies revealed evidences of moderate hypothyroidism. The administration of thyroid extract promptly relieved all symptoms.

COMMENT

With our present diagnostic aids it is difficult to demonstrate dysfunction of the cricopharyngeus muscle. In the presence of disease of the cervical esophagus such as carcinoma or foreign body and in chronic hypopharyngitis or pulsion diverticulum of the pharynx, it is reasonable to assume that incoordination of the swallowing function can occur. It is not possible to differentiate between this and the changes produced by the disease itself. Further, it cannot be ascertained whether reflex stimuli are producing increased involuntary tonic contractions (spasm) of the cricopharyngeus or there is lacking the necessary stimulus to relax the normally contracted sphincter. In addition, the circular muscle fibers of the upper esophagus which are striated and receive practically the same innervation as the cricopharingeus probably participate in the mechanism of incoordination of the upper esophageal constrictor. One, therefore, should consider in the differential diagnosis carcinoma, foreign body, pulsion diverticulum, congenital or acquired stenosis, chronic hypopharyngitis, chronic esophagitis, cardiospasm, disturbances of the thyroid gland and a host of other conditions about the neck and upper mediastinum. Certain laryngeal disturbances also may give rise to indefinite symptoms referable to the swallowing act.

CONCLUSIONS

Cricopharyngeal spasm, also referred to by many other designations, comprises a large group of disorders of the upper orifice of the esophagus.

Dysphagia is one of the manifestations of tension which may be present in any of the neuroses.

Dysphagia is a common symptom of organic disease of the hypopharynx and esophagus and also may be present in certain disorders of the larynx and periesophageal structures.

A diagnosis of cricopharyngeal spasm or other functional disorder should be made only after other diagnostic possibilities have been excluded by a thorough study.

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DISCUSSION

H. G. BEATTY, Columbus, Ohio: Patients with disturbance in swallowing are classified into one of two groups: That in which the condition is due to an organic lesion, as Doctor Clerf has mentioned, and that in which the disturbance is functional in origin. I hesitate to make a diagnosis of hysteria in any patient who complains of difficulty in swallowing until after esophagoscopy has been performed. Those who are familiar with the use of the esophagoscope know how often it has proven of value in locating an unsuspected malignant lesion.

There is one other cause for a reaction in the structures which may result in difficulty in swallowing, and that is allergy in which edematous obstruction of the esophagus occurs. The cricopharyngeus very often is affected. This should not be confused with spasm, because the edematous reaction can occur quite suddenly and not last too long.

I appreciate very much the opportunity of discussing this paper.

SAMUEL IGLAUER, M.D., Cincinnati, Ohio: I am pleased to discuss the subject of spasm of the upper end of the esophagus, and I think we are indebted to Doctor Clerf for emphasizing this symptom complex. Cardiospasm is assumed to be due to a spasm of the cardiac orifice, but much stress is not placed on spasm which occurs at the upper end of the esophagus. Doctor Clerf's paper concerns spasm of the cricopharyngeus muscle. Anatomically, this muscle is not a true sphincter. Two thirds of its circumference is composed of muscle, and the remainder is composed of cartilage. A sphincter is described as a ring-like muscle which closes a natural orifice of the body and in that sense the cricopharyn-the presence of carcinoma.

Doctor Clerf has mentioned the work of Elze, and Elze insists that the true sphincter in the upper end of the esophagus consists of an encirclement of muscular fibers. In visualizing these structures through an esophagoscope a stellate closure is seen which completely encircles the upper part of the esophagus. It is composed of muscular fibers. Therefore, I believe that this structure in the upper end of the esophagus and not the cricopharyngeus muscle is the factor which contributes to spasm in this region. This part of the esophagus is always in a state of contraction and when an instrument is introduced it may be necessary to wait until the muscle relaxes, or as the essayist has said, a general anesthetic may be given or cocaine applied to relax this tonic contracture.

Elze thinks that the fold above the real sphincter is due to a venous plexus which he has dissected out very carefully. When this is filled with blood it will produce a fold immediately above the stellate orifice. This region is also innervated by the recurrent laryngeal nerve. Therefore, as Doctor Clerf has said, sometimes a spasm of the esophagus is associated with disease of the larynx.

Doctor Clerf has also referred to the fact that ordinarily there is no ballooning of the esophagus in a patient who has had his larynx removed, and it occurred to me that probably the so-called "pharyngo-esophageal voice" after laryngectomy is due to a control which the patient has obtained over his sphincter.

Doctor Clerf cited several cases in which the diagnosis was not plain. I should like to mention one case. A woman, aged 62 years, stated that for five or six years she had been having trouble in swallowing and some pain on swallowing. She complained of a tight feeling in her throat. Physical examination revealed that she was rather anemic. Bilateral oophorectomy had been performed ten years before. Considering that the symptoms had been present for six years and the roentgenographic examination of the esophagus at the time she consulted me revealed normal findings, I believed that I was dealing with a functional disturbance. I dilated the esophagus with a mercury bougie two or three times, with no benefit to the patient. I did not see her again for seven months. At that time examination revealed a small lump in the neck anterior to the sternocleidal region. This was removed for biopsy, and it was found to contain carcinomatous tissue. Roentgenographic examination and hypopharyngoscopy revealed the presence of carcinoma.

I think that in this case almost anyone might have been deceived, because the history of difficulty in swallowing of six years' duration would have ruled out the probability of carcinoma. However, a patient with a functional disorder may develop carcinoma and occasionally does. In fact, it seems someone has suggested that cancer might originate in certain regions of the body following prolonged functional disorders in that region.

H. P. Mosher, M.D., Marblehead, Massachusetts: My autonomic nervous system has been actively stimulated by the paper and even more so by some of the discussion.

In the early days of esophagoscopy we had spasm of the lower end of the esophagus and spasm of the upper end. In our part of the country, we soon did away with spasm of the upper end of the esophagus. A great majority of the cases of globus hystericus proved to be due to webs in the pyriformis sinus or webs back of the cricoid cartilage. Later, after many years of investigation, spasm of the lower end of the esophagus, so-called cardiospasm, disappeared as a diagnosis with us and it has disappeared in certain other enlightened medical centers.

As I said, in Boston a diagnosis of globus hystericus does not appear. I can't remember when a case has been sent in with this diagnosis. Doctor Clerf's paper shows that the ghost of the spasm in connection with the esophagus still walks. The other day I asked Doctor Macmillan, our roentgenologist, how often he sees spasm of the cricopharyngeus. His answer was, "Practically never." He did say that of late he has seen what he calls hypertrophy of the cricopharyngeus and that the hypertrophy gave symptoms of difficulty in swallowing liquids, but no difficulty in swallowing solids, and the x-ray picture revealed apparent enlargement of the cricopharyngeus.

Some years ago a Frenchman published an article on acute spasm of the cricopharyngeus. He reported a number of cases. The condition was associated with infection, that is, with a sore throat. It lasted about three weeks. He classified them as spasm due to irritation from the infectious agent. For years, or ever since that paper came out, I have been hunting for that form of spasm, but I have never been able to see it.

I was interested in what Doctor Clerf said about the circular muscles of the esophagus taking up the work of the cricopharyngeus. This seems to me to be very possible.

I was interested also in what he said about the difficulty in examining the upper end of the esophagus, and the other discussers have said the same thing. For once I am in entire agreement with all the discussants. It is the hardest part of the esophagus to examine. Now we do not see spasm of the upper end of the esophagus. Put that down to lack of technic or what you will. Doctor Clerf reported a case of disturbance of the superior sympathetic associated with spasm of the cricopharyngeus. I do not deny that there are a few such cases, but by and large, we rarely see them. I feel that in the majority of these cases there is a web of some sort and my concluding point is this: In order to have your examination worth anything, the esophagus should be examined with as large a tube as can safely be used on the patient and the examination should be carried out under a general anesthetic so that whatever is there can be clearly visualized.

J. Vernal Cassady, M.D., South Bend, Indiana: I hesitate to follow Doctor Mosher, but I should have hesitated much more to precede him.

In a review of 92 cases of foreign body lodged in the esophagus, in 80 per cent of them the foreign body was lodged in the cervical esophagus just below the cricopharyngeus muscle. The pharynx squeezes the foreign body or food into the esophagus, forcing it through the cricopharyngeus and into the relaxed upper end of the esophagus. The muscular coat of the pharynx consists of three flat, thin muscles situated one above the other, known as the superior, middle and inferior pharyngeal constrictors. These muscles of the lateral and posterior walls have circular fibers which meet in the pharyngeal raphé. The inferior pharyngeal constrictor (slide) consists of two portions at its origin, the thyreopharyngeus and the cricopharyngeus, which blend into a single muscle after leaving their origin to surround the pharynx and be inserted into the median raphé.

The larger superior thyreopharyngeus arises from the surface of the plate of the thyroid cartilage. The inferior cricopharyngeal portion arises from the outer surface of the ring of the cricoid cartilage. The fibers of the two portions blend to form the inferior pharyngeal constrictor, the superior fibers running obliquely upward, the uppermost rather sharply while the cricopharyngeal portion runs almost horizontal. The median raphé which receives the insertion of almost all the pharyngeal constrictors is absent below so the lower fibers are circular below and continuous with the circular esophageal fibers.

These lower circular fibers, being attached anteriorly to the cricoid cartilage, pull the lamina backward, closing the mouth of the esophagus and act like a pinchcock on a burette shutting a rubber tube. Esophagologists too frequently regard the cricopharyngeus as a distinct muscle because of its prominence. In reality the cricopharyngeal fold of the inferior constrictor of the pharynx is only such a prominent fold because the lamina of the cricoid is pulled backward to close the lower

pharynx over the relaxed cervical esophagus, commencing directly below the edge of the cricoid lamina. The softness of the esophageal wall is in marked contrast to the rigid cricoid cartilage and its cricopharyngeal pinchcock.

The tunica muscularis of the esophagus is composed of two layers, a circular inner layer and an outer longitudinal layer, the outer being stronger and more highly developed. At the upper end, the longitudinal fibers divide into two bands which encircle the esophagus to pass anteriorly around it and be inserted into a tendinous band at the upper border of the cricoid cartilage. The separation of these stout fibers just below the cricoid cartilage leaves a weak triangular defect in the posterior esophageal wall. Here only the circular esophageal fibers and the inferior constrictor horizontal fibers which join with it are present. The relaxed upper end of the esophagus in contrast to the tight cricopharyngeal pinchcock, the squeezing action of the pharyngeal constrictors as compared to the weak peristaltic wave of the circular esophageal muscle, the narrow lumen occasioned by the broad lamina of the cricoid pushing backward plus the redundant mucosa and collapsed lumen of the cervical esophagus together create the ideal site for lodgement of foreign bodies in the upper end of the esophagus.

I have seen four cases of traumatic ulcer causing cricopharyngeal spasm; 20 abrasions following a history of foreign body in the esophagus where no foreign body was found and 31 patients with spasm of the esophagus who underwent esophagoscopy and in whom only a spasm was found, some of which were associated with mouth infection, two with excessive use of tobacco and the remainder simply neurosis or globus hystericus.

LOUIS H. CLERF, M.D., Philadelphia: I wished to bring this subject to the attention of the members of the Academy because, as Doctor Mosher has stated, cricopharyngeal spasm is not common, if it does exist. If spasm is present, it probably is due to contraction of the circular muscle of the esophagus.

THE FIXATIONAL CORNEAL LIGHT REFLEXES AS AN AID IN BINOCULAR INVESTIGATION

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ARMY AIR BASE
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THE ESTIMATION of binocular imbalance based on objective examination of the eyes has been found unsatisfactory because inspection of the eyes in routine muscle examination is often difficult. The role of the corneal light reflex as an aid in clinical practice has not been sufficiently explored, and my aim in this paper will therefore be to show how much light reflexes can be utilized clinically to complement the familiar cover test.

First, the meaning of the fixational corneal light should be considered. For practical purposes it may be regarded as that constant position of the light reflex on the cornea when each eye is made to fix on a light and when the observer's eye is behind the testing light. It may not necessarily be on the center of the cornea (angle gamma) even though the macula assumes fixation; but it represents the normal anatomic position of the light reflex for that fixing eye. As such, it is to be distinguished from the more peripheral corneal or deviational reflex, seen in a squinting eye when the fellow eye is fixing.

With an ordinary flashlight one also notes whether binocular fixation is as satisfactory as monocular fixation. The examiner keeps his eye directly behind the test light and makes a mental note of the positions of the light reflexes on both corneae respectively. He then covers each eye alternately and compares binocular reflexes with those obtained by monocular fixation. If such light reflexes assume the same corneal positions on binocular fixation as they do on monocular fixation, it indicates the absence of deviation for that testing distance. Displacement of the light reflex from the fixation position in one eye when light is directed to both eyes indicates deviation in that eye. If the reflexes appear centered on binocular light stimulation, but displacement occurs in one eye on attempted monocular fixation by alternate cover test it suggests false macula.

If a fixational corneal light reflex as defined above is regarded as indicating normal macular projection, displacement of the light reflex from this fixing position would indicate image registration on an extra-foveal portion of the retina. If a light be directed on the cornea from the nasal side so as to produce a corneal reflex medially, image registration on the retina will be temporal. The explanation may be simplified by stating that the position of the corneal light reflex in relation to the fixing position indicates the direction in which the image is being projected. (Fig. 1.) If the corneal light reflex be nasal to the fixing position, image projection is also nasal.

In terms of binocular imbalance, the light reflex in a divergent squint would be nasal in the deviating eye or in a direction opposite to the deviation, and diplopia, if present, would be nasal or crossed to correspond to the nasal displacement of the corneal light reflex. When the reflex is displaced upwards and nasally while the other eye fixes

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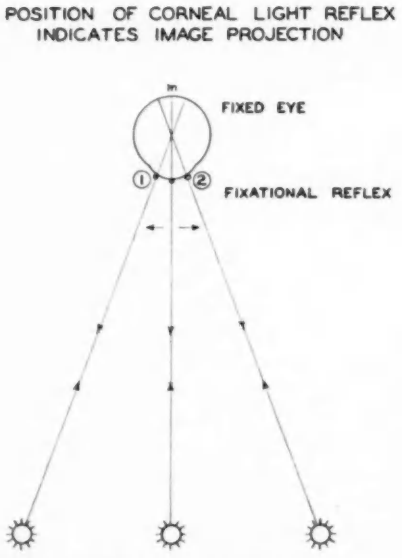
POSITION OF CORNEAL LIGHT REFLEX
INDICATES IMAGE PROJECTION
FIXED EYE
FIXATIONAL REFLEX
REFLEXES ① AND ② ARE PERIPHERAL AND
DIRECTION IN RELATION TO FIXATIONAL REFLEX
INDICATE PROJECTION OF IMAGE

FIG. 1 — Position of corneal light reflex indicates image projection.

(fixational reflex), it means a downward as well as an outward displacement of the eyeball (hypo-exotropia) and infero-temporal registration, and again an image projection upward and nasal corresponding to the displaced position of the corneal light reflex in relation to the fixing position.

To appreciate more fully the meaning of a corneal light reflex one should bear in mind the following principles:

a. The apical portion of the cornea is a convex spherical mirror and therefore the first or anterior corneal reflex has all the characteristics of an image produced by such a convex mirror.

b. The corneal light reflex is diminished in size, erect and virtual.

c. The construction of this image is shown in Fig. 2. Any ray incident through the center of curvature will return along the same path. An incident ray parallel to the principal axis will be reflected as coming from the focal point which is located one-half of the radius behind the surface.

d. Due to the relatively small radius of the cornea (about 8 mm.) the image plane for any test-light distance will practically coincide with the focal plane 4 mm. behind the surface. Even if the test light be at 333 mm. from the cornea the difference in location is only .05 mm.

e. The anterior or bright Purkinje corneal light reflex is, therefore, strictly speaking, not a superficial reflex, but retro-corneal, or 3-4 mm. behind the surface.

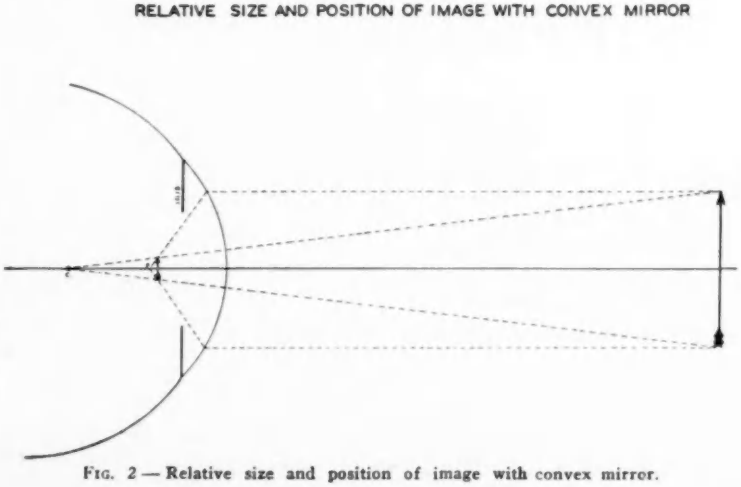

FIG. 2 — Relative size and position of image with convex mirror.

f. As a light is moved parallel to the facial plane of the patient the corneal light reflex moves in the same direction although to a much reduced extent.

g. The position of the corneal light reflex depends mainly upon the positions of the test-light relative to the principal axis upon the eye of the observer. The distance from the test-light to cornea is of no practical influence for reasons mentioned above.

It is important for purposes of accurate study to adopt a uniform procedure in studying corneal light reflexes. The position of the examiner's eye behind the light source is found to be most reliable and uniform.

If fixational corneal light reflexes are assumed to indicate an absence of deviation of the eye, it may be conceived that any artificial means of centering a peripheral corneal light reflex, as in squint, might likewise serve as a basis for measuring ocular deviation. In the case of the synoptophore the carriers are rotated until they are in direct line with the corresponding fixation positions on the corneae and readings are made. This is a far more satisfactory method than the crude Hirschberg test based on translating roughly the position of the corneal light reflex in a squinting eye in terms of millimeter displacement from the fixing position.

The peripheral corneal light reflex method (Hirschberg) is unsatisfactory for the measurement of squint because:

1. It is based on a rigid corneal curvature of 8 mm. which must vary in different persons.

2. It is difficult to control the patient sufficiently to measure the position of the light reflex accurately. Moreover, the position of the corneal light reflex changes with movement of the examiner's eye.

3. The amount of displacement measured in millimeters can be made only roughly and is never found to be uniform when tested by different examiners. Moreover, a displacement of only one mm. means 7 degrees, and one is in no position to make fractional readings.

4. It does not take into account the center of rotation of the eyeball in addition to the center of curvature of the cornea.

5. The corneal curve is not a geometric curve because flattening towards the limbus takes place.

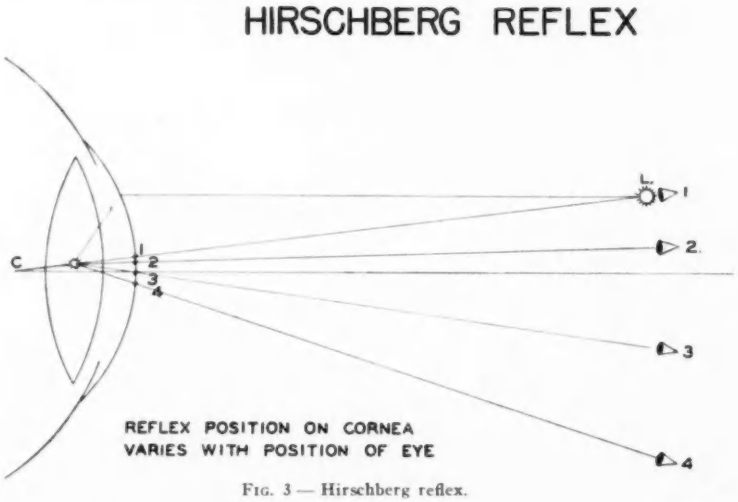

FIG. 3 — Hirschberg reflex.

6. The corneal light reflex as seen peripherally is distorted; whereas one seen at the center of the cornea is sharp.

Artificial restoration of fixational corneal light reflexes for measuring ocular deviation may be obtained as follows:

1. By Ordinary Flashlight

In convergent squint, the light reflex on the non-fixing eye will be displaced temporally from the corneal fixation point. As the test light is moved along a rule gradually closer to the eyes the light reflexes reach the corneal fixation points and on the basis of the normal primary deviation for that subject and the distance from the eyes (Pcb) one can calculate readily the amount of squint. In actual practice, however, this method of testing convergent squint is found wanting. One must be careful to stop just at the position at which the lights appear centered, otherwise an added convergence impulse may upset one's reading; P.D. measurement in convergent squint may prove difficult; and, finally, the readings obtained by such a method can be interpreted as deviation for that distance only and not for 20-foot or 13-inch test distances.

2. By Means of a Partition or Septum and Individual Test Lights at a Set Distance Between the Eyes

A light on one side of the septum is set in front of the fixing eye while the light on the other side for non-fixing eye is moved laterally or vertically or both until it too produces a light reflex on the corneal fixation point. When such position is reached alternate fixation to light stimulation should produce no eye movement unless amblyopia is present in the non-fixing eye. The examiner's eye should be behind the test light.

A septum placed between the eyes serves the purpose of a cover, but with the difference that both eyes can be observed at the same time. In orthophoria both eyes project to a common point on cover test. By moving the cover into a septal or between-the-eyes position, fixa-

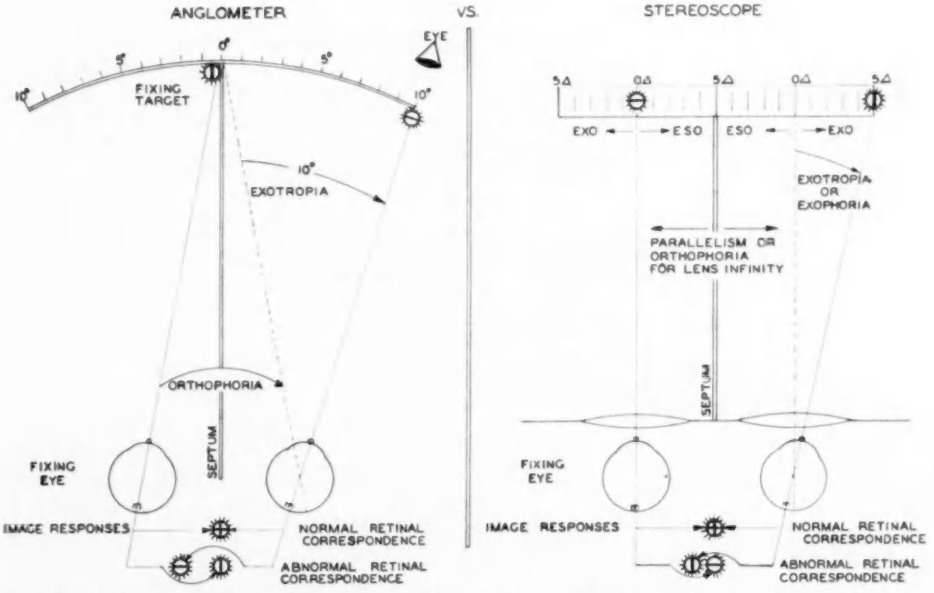

FIG. 4 — Anglometer vs. Stereoscope. Showing positions of targets in relation to ocular deviation, fixational corneal light reflexes and image responses.

tion by one eye on a corresponding light placed close to the septal wall will make the fellow eye converge in the direction of that unseen light in the case of orthophoria. In exophoria or exotropia the non-fixing eye turns out and the test light is displaced laterally to reach the corresponding corneal fixation point. In esophoria or esotropia the light reflex on the deviating eye is displaced lateral to the fixation point and supplementary base-out prism is required to center the light reflex in the deviating eye. (Fig. 4.)

The septum arrangement is of distinct advantage not only as a quantitative cover test, but also as a comprehensive means of comparing objective and subjective responses, notably in cases of abnormal retinal correspondence. When the images appear to touch or to correspond at the angle of deviation it speaks for so-called normal retinal correspondence because the subjective or image angle corresponds with the objective or deviation angle.

3. By Means of Synoptophore or Stereoscope

The synoptophore and the stereoscope are essentially devices which utilize the principle of the septum or independent stimulation to each eye. They also incorporate plus lenses to relax accommodation and convergence, thus bringing parallelism of the visual axes to within a comprehensive range. Whereas without the septum both eyes normally tend to converge on opposite sides of the septum, the incorporation of plus viewing lenses tends to shift the visual axes outward.

The basis for centering of the corneal light reflexes with the synoptophore appears quite simple. By rotating each carrier around a pivotal point the mirrors are so rotated as to bring the corneal light reflexes to their respective fixing positions at the angle of deviation.

With the Brewster stereoscope a critical corneal light reflex can likewise be obtained by employing a bright transparent target that does not cover too large a retinal field. As these targets are placed in the calculated primary positions for different accommodations one may readily note whether centering of the corneal light reflexes is maintained. The angle through which such targets are displaced medially or laterally relative to such primary positions in order to strike the centers or fixation points of both corneae serves as a measure of the amount of ocular deviation for that testing distance. The anglometer, also incorporating a septum, provides for studies of the corneal light reflexes at but one accommodative range (3 diopters) or a fixed distance of 13 inches; its flexibility lies in permitting one to study the positions and visual responses of the eyes in the different motor fields. The author's stereoscope permits one to study the corneal light reflexes in a single motor field or the eyes-front gaze but its flexibility lies in enabling one to study the corneal light reflexes at different viewing distances or accommodative ranges.

4. With the Aid of a Prism — THE PRISM-REFLEX TEST

In the routine study of ocular deviation a prism is used to nullify ocular motion on alternate covering. The role or the variations in the position of the corneal light reflex as a result of the prism has been largely overlooked, possibly for the reason that test targets set at a 20-foot distance are too faint to yield a visible corneal light reflex. In studying the effects of a prism with a test light at a 13 inch reading distance I found that whereas a ray of light traversing such a prism was refracted towards the base, reflection of light on the cornea was toward the apex. I found moreover that a prism sufficient to stop motion of the eyes on alternate cover

also had the fortunate property of centering the corneal light reflexes. This interesting phenomenon, to my knowledge, has not been reported previously in the medical literature. So here there is additional proof that whatever method one employs the primary aim in measuring latent or manifest squint is, first, to produce artificial restoration of corneal light reflex from a peripheral to a fixing position; and with prisms to ascertain strength required artificially to center these reflexes.

One can observe easily the change in position of the corneal light reflex with gradual increase in prism by experimenting with a glass eye and rotary prism. The light is directed to the center of pupil and both the observer's eye and test light are kept in line in a steady position. As prism is rotated either way it will be noted that light reflex moves in the direction of the apex of the prism and slight increase in prism strength will produce appreciable displacement of light reflex. The eye itself will also be seen to move towards apex of prism, but such gross change in apparent position of eyeball is distinct from that of the corneal light reflex because it is on a different image plane. One can simulate deviation by turning the glass eye in or out in relation to the test light which remains in the same position. When the eye is turned out, the light reflex is seen to move in from the fixation or central position. To bring the light reflex back to the fixation position the prism is merely set with apex in the direction of the center of the pupil. If the setting of the prism is compared to the direction which an arrow must take to reach the center of the pupil it simplifies matters.

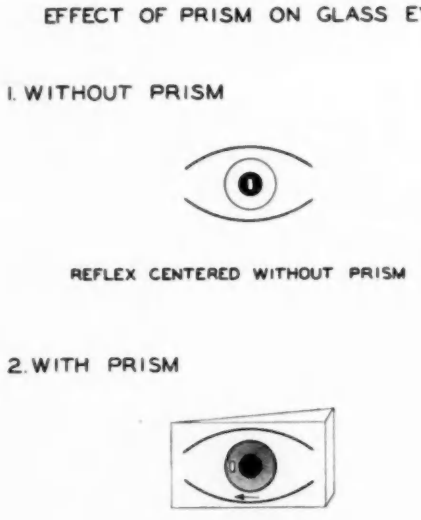
EFFECT OF PRISM ON GLASS EYE
1. WITHOUT PRISM
REFLEX CENTERED WITHOUT PRISM
2. WITH PRISM
LIGHT REFLEX DISPLACED TOWARDS APEX OR IN DIRECTION OF ARROW ALTHOUGH LIGHT SOURCE REMAINS FIXED.

FIG. 5 — Effect of prism on glass eye.

The prism reflex test simplifies the measurement of ocular deviation. The novice confronted with a case of squint may be at a loss to know how to place his prisms in order to neutralize the deviation. If he thinks in terms of fixational corneal light reflexes, he merely turns the prism first one way and then the other and notes the direction and amount of prism (using rotary prism) required to bring the light reflex in the deviated eye to a central position. In esotropia the eye is turned in and the light reflex is displaced temporarily; and a base-out prism centers the corneal light reflexes accordingly. If the eye turns upwards as well as inwards, the light reflex in the deviated eye is displaced downwards as well as outwards and both a vertical (base-down) as well as a base-out prism bring the light to the center of the cornea in that eye.

Certain precautions should be observed:

a. This test is most satisfactory for a 13-inch test range. For greater distance, a stronger light source must be employed to yield a bright corneal light reflex.

b. A rotary prism is the most satisfactory instrument for this test. A phor-

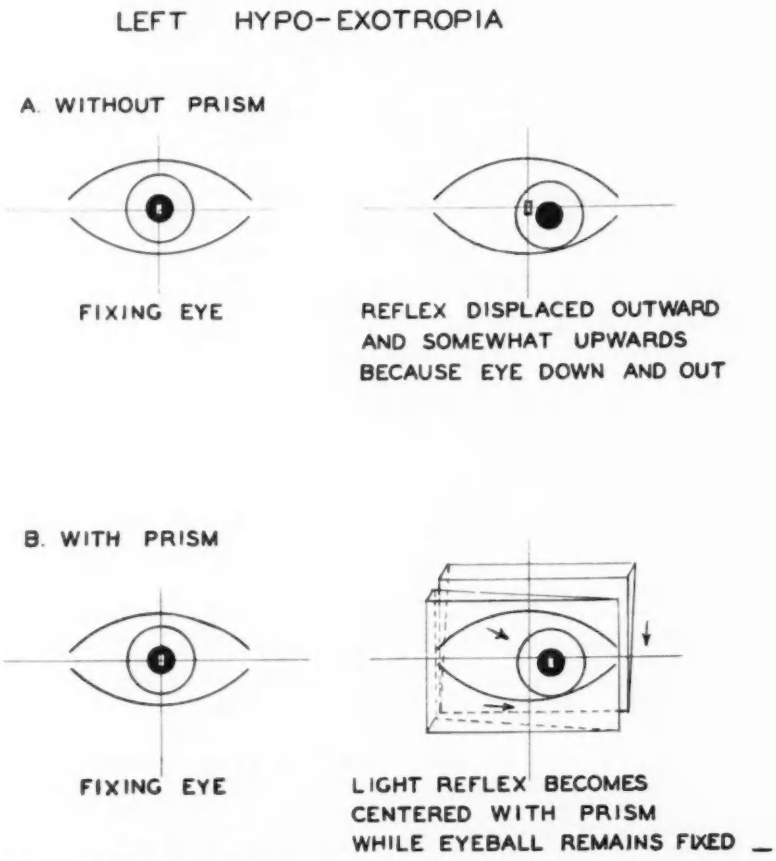

FIG. 6 — Hypo-exotropia. Showing restoration of fixational corneal light reflexes by prisms.

ometer is generally unsatisfactory because the framework covers the eyes in such a way that careful inspection of the reflexes is difficult.

c. As a rule the prism is placed over the fixing eye, and changes in the position of the corneal light reflex are observed in the deviating eye.

If in a case of manifest squint, the corneal reflex is displaced only laterally (horizontal squint), it simplifies prism testing in that a rotary prism alone is sufficient to produce centering of the corneal light reflexes. If the displacement be oblique, in other words, the squint is vertical as well as horizontal, the vertical component is first corrected by using a vertical prism, always remembering that the light reflex will shift in the direction of the apex of the prism. Specifically, if the reflex in the deviated eye is higher (hypotropia) the reflexes are first leveled by placing a prism with apex down. As a rule the vertical component requires much less prism than the horizontal component and when this correction is disposed of, one is in a position to neutralize the horizontal component with rotary prism placed in front of the vertical prism. The rotary prism is rotated so that light reflex moves in direction of apex of prism. If the reflex in the deviated eye is displaced medially (exotropia) the prism is rotated with

apex outward. After what appears to be centering of light reflexes in both corneae has been obtained, the familiar cover test is supplemented to ascertain whether ocular motion has been stopped and whether light reflexes remain steady on alternate covering. It often happens that this rapid preliminary prism-reflex test conforms quite closely with the cover test. In other instances, just a little more turning of the prism in either direction is sufficient to stop such movement. At any rate, one sees the eyes and their reflexes much more clearly than by relying on ocular movement alone.

The prism-reflex test does not replace the familiar cover test but simplifies it by permitting rapid inspection of the restoration of the corneal light reflexes to their fixation positions. The cover test alone is not infallible, for if one has to rely on gross appearance of the eyes alone he may be at a loss to tell exactly when movement is stopped. It requires a critical eye to judge cessation of fine ocular movements, and the prism-reflex test proves quite helpful as a rapid confirmatory test. Also, the cover test proves deceptive in certain types of manifest squint associated with false macula and in which there is no movement of the eyes on alternate covering. Here, the prism-reflex test offers a satisfactory estimate of the amount of squint.

In the placement of the prism, as a rule the examiner selects one eye for prism placement. I usually place the prism before the right eye. However, in marked squint, especially with amblyopia, the prism is set before the normal or fixing eye and then by rotating it in the direction of neutralization the corneal reflex is observed to become gradually centered in the otherwise deviating eye because the fixing eye moves in the direction of the apex of the prism. For this test I prefer the simple rotary prism to the bulky phorometer because it covers one eye rather than both, it is free from obstructing framework, it can be used on either eye and it can be used to study vergence as well as deviational response. It is also preferable to individual square prisms.

For excessive deviation no prism is reliable, and the angle method as embodied in the cardinal anglometer is the method of choice. The anglometer enables one to study deviations in all selected positions of gaze, and it has certain controlled features which further render such readings reliable. Moreover, it is based on physiological and optical principles which make it as sound as is feasible for clinical requirements.

CLINICAL APPLICATIONS OF THE CORNEAL LIGHT REFLEX TEST

1. Pseudo Squint—epicanthus; Angle Gamma
2. False Macula and Amblyopia
3. Abnormal Retinal Correspondence
4. Ocular Torticollis
5. Cardinal Positions of the Eyes
6. The Fixing Eye
7. Primary and Secondary Deviations
8. Prism Convergence and Divergence
9. Objective Heterophoria
10. Phoria vs. Tropia
11. Squint Measurement
12. Malingering

1. *Pseudo-Squint — Angle Gamma; Epicanthus*

In studying the corneal light reflexes, one first determines their positions on each eye with monocular fixation. Whether such reflex is found centered or somewhat off center (angle gamma), for practical purposes this is termed the corneal fixation point because it corresponds to macular fixation. In fact, the measurement of angle gamma may be disregarded and ocular deviation may be considered as the displacement of the corneal light reflex from the fixed corneal position assumed on monocular fixation. With both eyes open, binocular fixation would therefore imply the sim-

ultaneous registration of the corneal light reflexes on these monocularly fixating corneal points.

Epicanthus. By considering epicanthus in terms of corneal light reflexes, the absence of true squint is noted when such reflexes are found centered both monocularly and binocularly.

2. *False Macula and Amblyopia*

False macula as distinguished from abnormal retinal correspondence is a form of amblyopia in which monocular foveal fixation is faulty. The corneal light reflex is thus seen to waver as the eye searches over a paramacular area to register imperfectly what the fovea is unable to accomplish. Fixation is unsteady and the position of the corneal light reflex is not uniform. As a rule, it is displaced temporally in convergent squint and nasally in divergent squint. It may be confused with angle gamma when the light reflex is not centered, but the nature of such fixation should serve to distinguish these two conditions. In some cases the corneal light reflexes appear centered when both eyes are open, but alternate covering will show the nature of monocular fixation and reveal the existence of false macula. In other instances the light reflex may appear centered on attempted monocular fixation whereas foveal fixation would yield a gamma corneal reflex.

In false macula with visible deviation in the amblyopic eye, one often finds that the eyes fail to respond to alternate covering and the poor eye remains deviated both with and without cover. In such cases the corneal light reflex test (with prism) proves quite satisfactory for measuring the deviations. In convergent squint a base-out prism placed over the good eye will make that eye turn in in order to fix the light; and the poor eye will therefore turn out until the light reflex will be found centered on that cornea as well.

While one cannot judge visual acuity objectively by observing the corneal light reflex, one can with a little experience suspect amblyopia on the basis of the relative unsteadiness of the corneal light reflex in the amblyopic eye. This test is not infallible, but it is occasionally helpful especially when dealing with cases of malingering.

3. *Abnormal Retinal Correspondence*

Abnormal retinal correspondence should not be confused with so-called false macula. In abnormal retinal correspondence monocular fixation in either eye is not disturbed. However, a perverted relationship exists between the macula of one eye and the paramacula of the other whereby diplopia is avoided. This relationship is interchangeable and exists with either eye assuming the role of fixation.

Abnormal retinal correspondence is a frequent manifestation of manifest squint. It differs from normal retinal correspondence (see Fig. 4) in that bimacular fixation or fusion or superposition of images does not occur when bimacular stimulation is made possible through artificial means, as by prism, stereoscope, or anglometer. One can detect abnormal retinal correspondence with any device which provides independent and simultaneous light stimulation to both eyes sufficiently strong to produce satisfactory corneal light reflexes. Measurement is based on moving the lighted targets along a calibrated arc (as in the anglometer) or graduated bar (as in the stereoscope) until they are in line with the respective fixing positions of both corneae. After noting this objective angle on the basis of such artificial centering of the corneal light reflexes and subsequent cessation of ocular movement on alternate flashing, the patient notes whether the images appear to touch. Approximation of these independent images at this deviation or objective angle denotes normal retinal cor-

respondence. Appreciable displacement of these images suggests an abnormal response or so-called abnormal retinal correspondence; and the lighted targets are again moved until their images approximate or cross at this new or subjective angle, and readings are again made. Thus in exotropia the images would appear to coincide not at the angle of the squint but at some angle nearer the "O" or primary position; likewise in esotropia the images appear to coincide at a less convergent angle than the deviation angle.

4. *Ocular Torticollis*

Tilting of the head in normal instances does not result in binocular imbalance or diplopia. In spite of such positional changes of the head, the corneal light reflexes will remain centered. This objective manifestation of normal binocular responses proves additionally helpful in interpreting whether a head-tilt is ocular or orthopedic in origin.

If in a case of habitual head-tilt the corneal light reflexes remain centered when the head is straightened, it suggests that such a head tilt is not ocular in origin. In ocular torticollis, the reflexes are centered only in the position where the eyes can see binocularly. In other positions of the head, displacement of the light reflex occurs in the non-fixing eye and the position of this displaced light reflex in relation to the fixing corneal position suggests the nature of the diplopia and the character of the deviation.

5. *Cardinal Positions of the Eyes*

Due to the asymmetric conformation of the upper lids one is never certain from a cursory inspection of the eyes in the oblique fields of gaze whether or not a paralysis of a levator or a depressor muscle exists. Alternate covering for these oblique fields may not prove feasible especially in an uncooperative patient. However, in the corneal light reflex test one is often surprised to learn

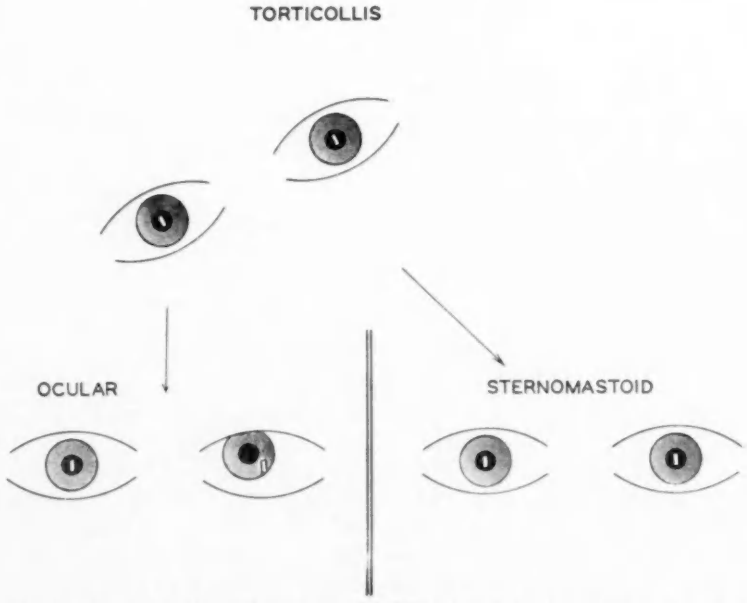

FIG. 7 — Ocular vs. Sternocleidomastoid torticollis in relation to positions of corneal light reflexes.

at a glance that what may appear to be a lag or paralysis of the superior rectus proves to be a false alarm due to this uneven conformation of the lids. This pseudo squint is analogous to the epicanthus simulating convergent squint.

In routine testing with an ordinary flashlight, movement of the examiner's eye for testing in the oblique fields proves quite clumsy, because his eye must keep moving behind the flashlight to attain uniformity in response. However, in the Bielschowsky method the head is tilted in different positions to produce the effects of oblique gaze of the eyes, and the examiner can maintain his head in a uniform position by operating test light from this set position.

The cardinal anglometer lends itself quite satisfactorily to controlled measurements and inspections of the light reflexes for definite positions of gaze. Some patients may not be in a position to move their eyes obliquely upwards or downwards for more than but a few degrees. It makes a difference if one's readings are based on an upward gaze of 30 degrees or of 10 degrees. With the anglometer one is in a position to note accurately the variations in imbalance with different selected positions of gaze, as well as the limits of oblique gaze. The head is kept fixed in a head-rest so as to prevent instinctive turning with movements of the eyes. Measurements can be made in but a fraction of the time required to make crude readings by prisms, and the test distance is fixed at 13 inches in all directions of gaze. With prisms one is never certain of his operating distance; instinctive head-tilt cannot be properly controlled, and multiple prisms are not simple to manipulate. Also, in marked deviation the reliability of prism readings drops sharply whereas angle readings conform to the angular deviations of the eyes along an arc.

6. Fixing Eye

When a test light is brought gradually closer to the eyes so as to elicit convergence, a point is reached when convergence is "broken" and one eye turns out and its reflex becomes displaced nasally. This is the non-fixing eye. When the corneal light reflex assumes a different position on alternate monocular fixation (with cover test) as compared to binocular stimulation, it may be assumed that the eye which maintains the same position of the corneal light reflex both binocularly and monocularly would be the fixing eye.

7. Convergence Near-Point

When a light is brought gradually nearer to the eyes a convergence impulse is brought automatically into play and in terms of corneal light reflexes the proximal range is determined at which such fixational corneal light reflexes can be maintained, or convergence near-

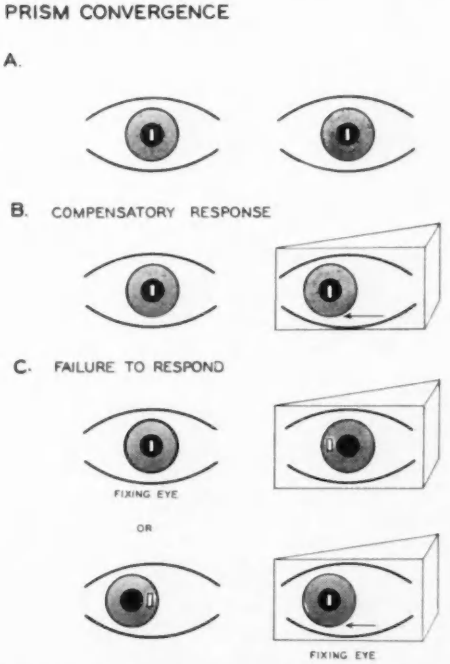

FIG. 8 — Prism convergence with maintenance of fixational corneal light reflexes.

point. It is also the distance at which reflex displacement occurs on one cornea. It is a simple objective test, and the examiner can note the "break" and foretell the nature of the diplopia.

8. *Prism Convergence and Prism Divergence*

The amount of base-in or base-out prism which one can overcome with rotary prism is a measure of the prism-divergence or prism-convergence. The prism-reflex test lends itself satisfactorily in revealing to what extent fixational corneal light reflexes can be maintained. A displacement in the light reflex from this fixation position as a result of increasing prism indicates a "break" in fusion and is of objective value in cases of suspected malingering when fixational reflexes are maintained in the presence of moderate strength of increasing prism.

In routine prism tests the patient is often relied upon to tell when fusion is "broken," and by overlooking the objective value of the prism-reflex test what appears as fusion or prism compensation is in reality relative suppression in one eye. In such cases, the light reflex will keep shifting in the non-fixing eye as prism is increased until stimulation of a more sensitive zone beyond the suppression area produces diplopia.

9. *Objective Heterophoria*

A prism (say 10 prism diopters base-down) placed vertically over one eye causes vertical diplopia ordinarily. The nature of such vertical diplopia depends on whether an orthophoria, an esophoria, or an exophoria is being dealt with. (Fig. 9.) In orthophoria the position of the eye is not changed as a result of such vertical prism, and the light reflex is displaced vertically or in the direction of the apex of the prism. If the eye behind the prism be the non-fixing eye, the light reflex will be higher and the false image created by that prism will also be higher than the image seen by the fellow or

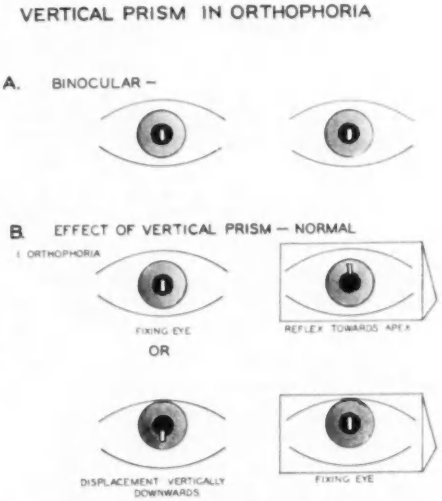

FIG. 9 — Vertical prism in orthophoria. Showing displacement of light reflex towards apex of prism.

fixing eye. If the patient transfers fixation to the higher image by turning the eyes upward, the light reflex becomes centered in the eye behind the prism and correspondingly lowered in the formerly fixing eye. The displacement of the light reflex in either eye suggests diplopia especially when alternate vertical fixation occurs. The nature of such light reflex displacement indicates the type of diplopia as well as the nature of the phoria.

In esophoria (Fig. 10), the non-fixing eye turns in behind the vertical prism, and in addition to vertical displacement of the corneal light reflex towards apex by this prism there is an added temporal displacement of the corneal light reflex as a result of the turning-in of the eye. Here too the position of this displaced light reflex indicates the nature of the false projection and incidentally of the type of heterophoria. From the standpoint of measurement that amount of lateral prism which will bring the light reflex into a straight vertical position or in an orthophoric position also serves as a measure of the amount of esophoria. By such vertical realignment of these

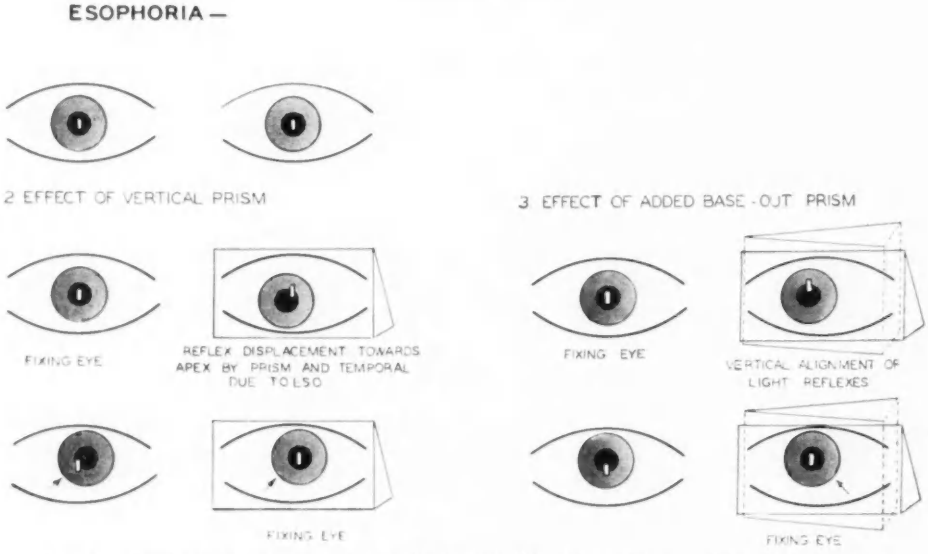
ESOPHORIA —
2 EFFECT OF VERTICAL PRISM
FIXING EYE
REFLEX DISPLACEMENT TOWARDS APEX BY PRISM AND TEMPORAL DUE TO LSO
FIXING EYE
3 EFFECT OF ADDED BASE-OUT PRISM
FIXING EYE
VERTICAL ALIGNMENT OF LIGHT REFLEXES
FIXING EYE

FIG. 10 — Esophoria and restoration of vertical corneal light reflexes by neutralizing base-out prism.

dual reflexes the images become likewise vertically realigned.

In exophoria, the non-fixing eye turns out behind the prism and the corneal light reflex is displaced inwards as a result of the phoria and upwards as a result of the vertical prism. Here again that amount of base-in prism required to restore vertical alignment of these dual light reflexes indicates the amount of exophoria. Also, the oblique positions of this displaced light reflex relative to this fixed position indicate the direction of projection of this false image.

10. *To Distinguish a Phoria from a Tropia*

In phoria as distinguished from tropia the eyes can respond to certain artificial stresses, such as by prisms, and still maintain binocular single vision and fixational corneal light reflexes. In tropia, the placement of a prism before the fixing eye will merely cause both eyes to turn in the direction of the apex of the prism (conjugate movement) in order for the fixing eye to maintain fixation behind prism, and the position of the corneal light reflex will change only in the non-fixing eye; the placement of the prism before the non-fixing or passive eye will merely serve to move the light reflex in that eye without altering the position or movement in either eye. In either case, that amount of prism required to neutralize the deviation will also serve to bring the light reflexes to the corneal fixation positions. In phoria,

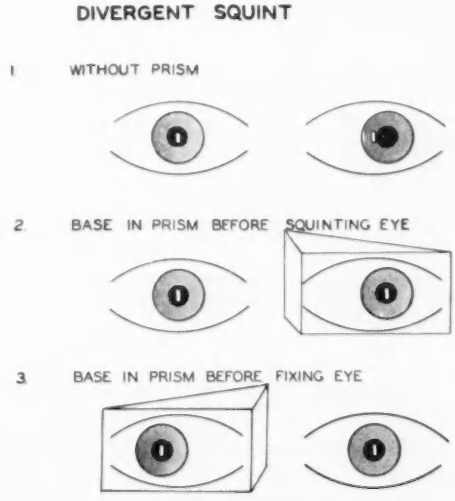

FIG. 11 — Divergent squint.

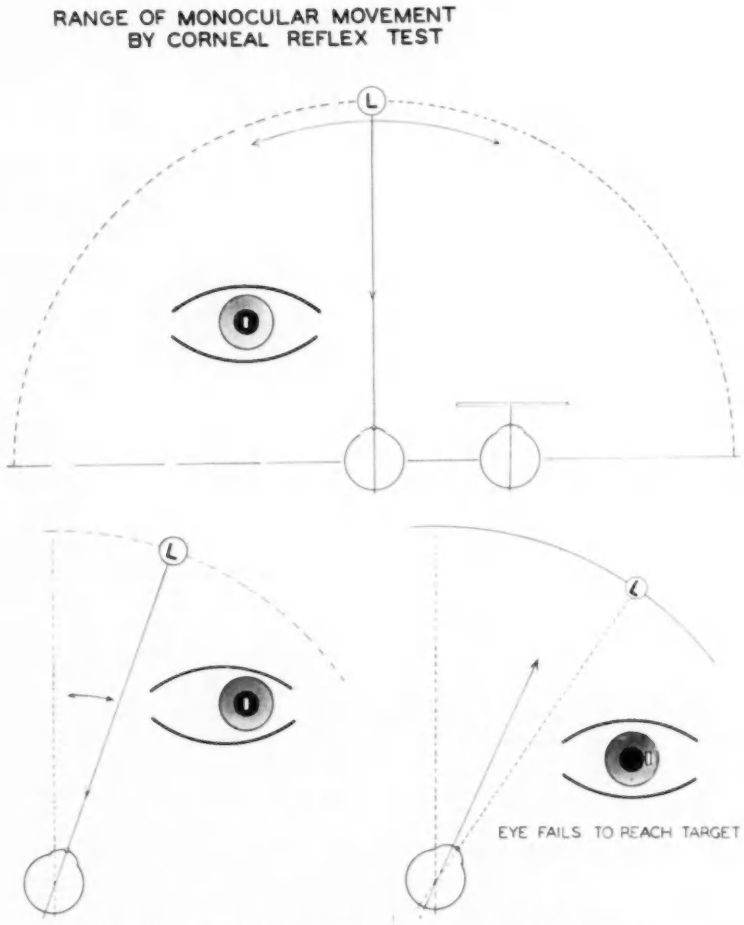

FIG. 12 — Range of monocular movement by corneal reflex test.

either eye will overcome by the same amount the effects of a prism and still maintain binocular single vision (fixational corneal light reflexes).

11. *Paralytic Squint — Primary and Secondary Deviations*

In paralytic squint the factors to be determined are (a) the fixing eye which has the fixational corneal light reflex, (b) the type and amount of displacement of the corneal light reflex in the fellow eye, (c) the changes in displacement of the light reflex in the non-fixing eye as the eyes are shifted in different cardinal directions of gaze, and (d) the amount of induced displacement in the otherwise fixing eye when the non-fixing eye is made to fix.

12. *Monocular Movements*

The central corneal light reflex test proves invaluable in measuring objectively the amount of monocular motility.

With the anglometer, the patient's head is placed firmly in a head-rest and the testing eye is brought into a cyclopean position (other eye covered). The lighted target is moved along the calibrated arc and the observer notes through what range fixation of the corneal light reflex can be maintained, first

in one direction and then in the other. The motility area can be plotted so as to include both the vertical as well as the horizontal directions of gaze.

CONCLUSIONS

1. While the cover test rightfully serves as a basis for estimating both latent and manifest deviations, it is not always easy for the examiner to observe slight movements in either eye on alternate cover.

2. The peripheral corneal light reflex test (Hirschberg) is an unsatisfactory objective test and is subject to the whims of the individual examiner.

3. Artificial restoration or centering of the corneal light reflexes to their fixation positions is a controlled and graded objective method for measuring ocular deviation and binocular imbalance.

4. Artificial centering of the corneal light reflexes can be obtained by acceptable methods which enable one to study binocular responses. Such methods include prism tests, anglometer, phorometric stereoscope, or synoptophore.

5. A prism refracts light towards the base, but displacement of the corneal light reflex takes place towards the apex. In squint a correcting prism is so placed as to shift the corneal light reflex to the fixation position on the cornea. That amount of prism required to center the corneal light reflexes also serves as a measure of the deviation.

6. The position of the peripheral corneal light reflex indicates the projection of the false image in case of diplopia. To overcome diplopia artificial centering of such peripheral corneal light reflexes is required. A prism placed with apex in the direction of the deviation will restore light reflexes to fixation positions.

7. The prism-reflex test simplifies the measurement of ocular deviation. It is intended to complement rather than to replace the familiar cover test, and is of objective value:

(a) In determining the amount of prism-convergence and prism-divergence as determined by the extent through which fixational corneal light reflexes can be maintained with increasing prism.

(b) As a measure of heterophoria.

(c) In distinguishing a phoria from a tropia.

(d) In measurement of squint with marked amblyopia.

(e) In malingering.

8. A rotary prism is the most satisfactory prism device for observing the gradual correction from peripheral to fixational corneal light reflexes in manifest squint.

9. The refined Brewster stereoscope enables one to center the corneal light reflexes at the angle of deviation. Moreover, it can be adapted to different ranges of accommodation.

10. The anglometer provides a comprehensive picture of both the horizontal and vertical deviations in any measured position of gaze through the incorporation of lighted carriers that can be adjusted to correspond to ocular deviations for those different positions.

11. The fixational corneal light reflexes prove additionally helpful in the objective study of:

(a) The Convergence Near-Point.

(b) The Fixing or Dominant Eye.

(c) Primary vs. Secondary Deviation.

(d) Ocular vs. Sternomastoid Torticollis.

(e) The Range of Monocular Movements.

(f) Binocular Fixation.

(g) Abnormal Retinal Correspondence.

(h) True vs. Pseudo-Squint (Angle Gamma).

(i) Amblyopia.

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CONGENITAL BLEPHAROPTOSIS — A CLASSIFICATION. THE PRINCIPLES OF SURGICAL CORRECTION

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PHILADELPHIA

"With an auspicious and a drooping eye."
HAMLET, I.ii.11. (Edition R. G. White)

FROM the standpoint of its correction, congenital ptosis still continues to be the step-child of ophthalmology. Not so long ago it shared this peculiar distinction with strabismus. In recent years, however, even the pediatrists, not to mention the family physicians, seem less certain that children outgrow a squint.

Congenital ptosis, in a large percentage of instances even when recognized, is still permitted to continue uncorrected, trusting that nature will work a miracle for the child. While hard to believe, it is a fact that in too many instances ptosis is not recognized before the child is 3 years old and even later in life. Histories obtained from parents prove this not infrequently. Diagnosis and treatment in the fourth year, the fifth year, or even the sixth year may still be early enough as far as end results are concerned, but when a 16 year old child of a physician reports for surgical correction with vision of 5/150, practically intractable amblyopia in that eye, then one is fully justified in thinking that carelessness is also a factor.

Congenital ptosis, excluding certain complicated cases, is a result of congenital developmental defects with and in the levator, the superior recti muscles and their innervations. There is a rather inconstant familial relationship to this. The cosmetic blemish alone of unilateral or bilateral ptosis of the upper lid or lids as the result of a simple impairment of the nerve that supplies the levator muscle should be sufficient to demand correction before the child develops amblyopia from impaired vision, awareness of the defect, shyness, reticence and inferiority complex.

Not infrequently paralysis of the superior rectus muscle, unilateral, homolateral, bilateral and contralateral, is at least of equal importance in the causation of the other complications of ptosis which are even more serious than the simple cosmetic defect. These complications include amblyopia, the loss or lack of development of single binocular vision, of curvature of the spine, epicanthus, torticollis, and/or spasm of the occipitofrontalis. These are situations which can and frequently do affect the child to a profound degree — even when mental adjustment or compensation is achieved. Occasionally the child fails to develop necessary mental and physical adjustments adequately. The result which we see in such instances is more than sufficient reason for that bit of sarcasm which opened this paper.

An analysis of private and clinical cases of blepharoptosis seen over the past 20 years shows 9 classes as a basis for the following classification of this condition, with the incidence percentage as well for each class.

Presented at the Forty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 11-14, 1942, Chicago, Illinois.

From the Graduate Hospital of the Graduate School of Medicine, the University of Pennsylvania, and from The Wills Hospital, Philadelphia, Pennsylvania.

Class 1. Unilateral ptosis without involvement of the superior rectus muscle. These included 7 cases with unilateral amblyopia of 6/22 or less; one case with torticollis. Forty per cent of all cases.

Class 2. Unilateral ptosis with involvement of the homolateral superior rectus muscle. These cases included 4 with amblyopia of 6/22 or less and 2 with torticollis. Twenty per cent of all cases.

Class 3. Bilateral ptosis without involvement of the superior rectus muscle. One case had a unilateral amblyopia of less than 6/22. Seven per cent of all cases.

Class 4. Bilateral ptosis with involvement of the superior rectus muscles. In 4 instances the involvement of the superior rectus muscle was one sided only; in 2 cases the patients had torticollis. Eight per cent of all cases.

Class 5. Unilateral ptosis with weakness of both superior recti muscles, which was more marked, however, in the homolateral eye. Two such cases have been seen. Both patients had torticollis and some degree of amblyopia. One patient maintained fixation usually with the eye which had the ptosis, i.e., the left eye. A secondary deviation of the right eye with fixation maintained in the left eye, because of better vision in the left eye, was the cause of the diplopia. The other patient had an almost complete ptosis. Two per cent of all cases.

Class 6. Ptosis with more or less complete third nerve and even sixth nerve paralyses; in 4 of these cases the condition was bilateral, and in 5 others it was unilateral; 8 of the cases showed torticollis; one case, a young lady, 20 years of age, in whom the ptosis was unilateral, had an amblyopia with vision of less than 1/100. Her degree and type of squint (divergent) was that of a strabismus fixus. Another patient in whom the ptosis was bilateral had possible inferior and superior rectus muscle paralyses. One patient in whom the ptosis was unilateral had involvement of the internal and the superior rectus muscles; a second had ptosis with impairment of the left internal rectus muscle and the third had impairment of the superior rectus muscle, homolateral, with fibrosis of both inferior recti muscles. Nine per cent of all cases.

Class 7. Ptosis with the classical jaw-winking reflex; the Marcus-Gunn syndrome of misdirection of developing fifth nerve, cranial nerve and tracts and the oculomotor nerve fibers.* Two per cent of all cases.

Class 8. Ptosis with the Duane's retraction syndrome. Both were undiagnosed until the patient was 10 years of age; at least the child had not been seen by a physician until then. Two per cent of all cases.

A patient whose case is not included had ptosis only when the left eye was rotated to the right and there was also a complete congenital sixth nerve pa-

* According to Bing and Haymaker (5th Ed., C. V. Mosby Co., 1939, pp. 589), "The Marcus-Gunn phenomenon occurs not only in cases of congenital ptosis, but also in individuals with acquired ptosis and in some who are free of ptosis. According to Helfreich and Bernhard, this phenomenon can be explained only on the basis of neuronal intercommunication between the nuclei of the facial, trigeminal and oculomotor nerves. Such an intercommunication has been recently denied by Lowey, Groff and Grant. These authors state that the Marcus-Gunn phenomenon is completely abolished by section of the motor root of the trigeminal nerve but that with forcible movements of jaw postoperatively the associated movement of the eyelid could be elicited. This suggested to them the presence of a proprioceptive reflex arc, an afferent limb of which was the sensory portion of the mandibular division of the trigeminal nerve. They assumed that the efferent part of the arc — that passing to the eyelids — was via autonomic fibers whose pathway was undermined but thought to be via the ophthalmic division of the trigeminal nerve." The author cannot agree with this hypothesis, considering the third nerve motor innervation of the levator, and judging from the many other not uncommon cranial nerve pathological intermingled syndromes seen, congenital as well as acquired. Nevertheless, the question is answered as unsatisfactorily by postulating the presence of brain-stem internuclear communications, as suggested by Helfreich and Bernhard.

ralysis on that side. The case does not represent a retraction syndrome (though enophthalmos is present) nor a strabismus fixus.

Class 9. Ptosis with neurofibromatosis. In 13 cases of orbital plexiform neurofibromata, ptosis was present in 10 instances to a marked degree, due to the neoplastic infiltration and to destruction of, or interference with, the levator. Ten per cent of all cases.

In an analysis of these cases, as to incidence, it is proper to omit those cases of neurofibromatosis, for they are not related in incidence, nor in cause. If this is done, then it will be found that in a few more than 100 cases of congenital ptosis, the following significant and interesting factors appear: Approximately 70 per cent are unilateral and 30 per cent bilateral. Of the cases in which the ptosis was unilateral, approximately 26 per cent were complicated situations, that is, other factors of clinical significance were present besides the cosmetic blemish of the drooping lids. In the cases of bilateral ptosis, almost all the patients had exaggerated epicanthal folds, especially the younger patients, and in addition to this, 37 per cent of the cases of bilateral ptosis were complicated by factors other than the epicanthus and the bilateral drooping of the lids. Epicanthus, alone, is a condition for which surgery should not be performed until the adult facial growth of the patient has been reached, for in many of these instances the condition corrects itself spontaneously with and when that growth is reached. So often this condition is more complicated than the average casual glance would suggest. The palpebral fissure is definitely shorter than normal, the caruncle is absent and both upper and lower lids are deficient in soft tissue in their horizontal levels. Crescentic excisions over the epicanthal folds simply put a drag upon the lids and the palpebral fissure without correcting the defect. Surgery, after adult growth is reached, means some type of flap correction and very often with it an external canthus plastic procedure to lengthen further the palebral fissure. The ptosis in these cases should always be corrected as early as is possible, and the epicanthus correction delayed for as long a time as is permissible. When the superior recti muscles are intact, their utilization for bilateral ptosis is ideal in that this technique modifies, in the least, the accompanying epicanthus. (See Fig. 1.) Instances may arise, however, when the degree of epicanthus is so severe that some corrective

FIG. 1 (a) — Unilateral ptosis without epicanthus. (b) Ptosis with definite epicanthus, dissimilar in degree in the two eyes.

surgery is indicated before that time (i.e., age of adult facial growth). There are some other congenital defects to be noted, as external canthal defects, ankyloblepharon, epiblepharon, primary epicanthus of high degree, lid colobomata, etc., which may have an accompanying ptosis. (See Figs. 2 and 3.) In these cases, however, this is only secondary to the plastic defect, and usually not of neuromuscular origin.

FIG. 3 — Congenital ptosis on left with lagophthalmos on right, upper and lower lids, both deficient. No disturbances of the extra-ocular muscles except for the levator on the left.

FIG. 2 — **Ptosis with congenital coloboma, upper lid. Bilateral ptosis with congenital colobomata, both lower lids.**

The following case reports illustrate certain of these subdivisions rather well:

CLASS I

Case 1. The patient was a young man 17 years of age with unilateral ptosis which had been present since birth. A paternal uncle was similarly affected. Examination revealed the vision in the right eye to be 6/30. (See Fig. 4.) The surgery carried out consisted of a levator advancement.

CLASS II

Case 2. This case represents unilateral ptosis, with unilateral paralysis of the homolateral superior rectus muscle. Following surgery, diplopia developed in the field of the left superior rectus muscle. Early surgery and the use of prisms since have maintained single binocular vision without the development of amblyopia from non-use. (See Fig. 5.) Surgery consisted of a Hunt-Tansley operation.

FIG. 4 — **Simple unilateral ptosis before and immediately after a levator advancement.**

FIG. 5 (a) Unilateral ptosis with unilateral paralysis of the superior rectus on left. (b) Condition one year later following Hunt-Tansley operation and superior rectus surgery. (c) Same case three years later; observe the level of the left eye, also the smooth surface folds of the upper lid on left.

CLASS III

Case 3. Bilateral ptosis is represented in Figure 6. A condition similar to that present in the patient existed in a younger sister, though in the case of the latter one eye showed a weakness of one of the superior recti muscles. The patient, a brilliant student in mineralogy, was definitely hindered in his laboratory work when using the microscope. Amblyopia was not present. The Shoemaker modification of the Motais operation was performed. (See Fig. 6.) Notice the presence of the occipitofrontalis spasm before surgery and the absence of this after surgery.

CLASS IV

Case 4. The patient manifested bilateral ptosis of high degree, with bilateral involvement of the superior rectus muscles, unequal in degree. There was practically complete paralysis of the superior rectus muscle of the left eye. Torticollis was present. At surgery some fibrosis of the left inferior rectus muscle was seen. Bilateral Hunt-Tansley operations were performed with advancement of both superior recti and recession of both inferior recti muscles. Further levator surgery may be carried out later on, if indicated. The patient had a marked occipitofrontalis

FIG. 6 (a) — Upper left, original condition. (b) Upper right, following Shoemaker modification of the Motais principle. Sutures still in position. (c) Lower left, same patient one year later. Notice the formation of lid folds and the absence of any occipitofrontalis spasm. (d) Lower right, same patient showing the degree of lid closure possible.

spasm and was developing a very definite curvature of the spine. The child presented a rather definite behavior problem as well. (See Fig. 7, lower figures.)

CLASS V

Case 5. The patient, a young man 25 years of age who was a student of divinity, was much handicapped as a result of unilateral amblyopia of moderate degree and torticollis, and, preceding any surgery, by diplopia. Surgery consisted of unilateral levator advancement, recession of the overacting right inferior oblique muscle, recession of the left inferior rectus muscle and advancement of the left superior rectus muscle. The end result revealed good function without diplopia. (See Figs. 8 and 9.)

inferior oblique muscle. Following surgery there was a most remarkable change in the child's behavior. Within 7 weeks, postoperatively, the child was an affable, happy, cooperative youngster. (See Fig. 10 A and B.) Figure 10 C illustrates ptosis on the left side with incomplete paralysis of the right third nerve. (Internal and inferior recti involved, OD.)

Figure 11 A illustrates the case of a girl, age 10, with bilateral ptosis; torticollis; a high degree of hyperopia; bilateral undulatoric, horizontal, ocular strabismus; divergent squint, alternating in fixation; disinclination for downward and upward rotations to a degree wherein both superior and inferior recti muscles were considered to be involved. A

FIG. 7 — Upper group, at age of one year, and one year later, all prior to surgery. Lower group (a) and (b): Patient six months prior to surgery, right and left, side views. Notice especially the torticollis present and the strong arching backward of the head and of the spine. (c) Same patient following a Hunt-Tansley procedure and the muscle surgery.

CLASS VI

Case 6. The patient, a child 6 years of age, had incomplete unilateral ptosis of the left eye, with incomplete paralysis of the third nerve on the left side and no internal rotation whatsoever. Accommodation and pupillary reflexes were intact. There was no amblyopia. The right facing and the torticollis indicated the patient's attempted escape from diplopia. The child was nervous, irritable and definitely unhappy. Surgery consisted of recession of the external rectus muscle and advancement with resection of the internal rectus muscle, left eye, and a Reese orbicularis procedure and tenotomy of the right

similar condition was present, though to a much less degree, in a brother 6 months old. Surgery consisted of bilateral advancements with resection of both internal recti muscles and bilateral levator advancements. Figure 11 B illustrates a case of ptosis with concomitant convergent squint. Figure 11, C and D, illustrates a case of ptosis with a paralytic external rectus muscle on the same side.

CLASS VII

Case 7. The patient, a young man 26 years of age, manifested a left jaw-winking reflex. (This is one of two such cases. However, photographs were not available of either pa-

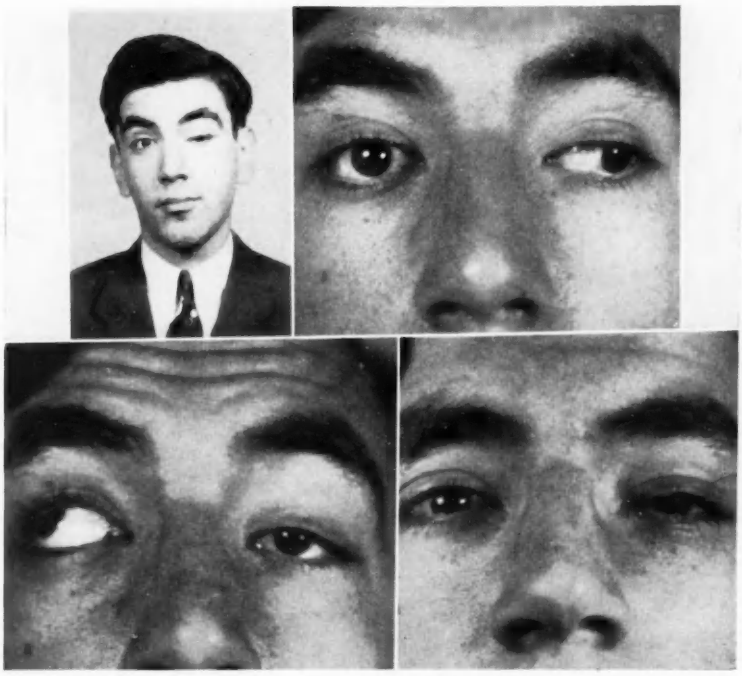


FIG. 8 (a) — Condition one year prior to surgery. Torticollis, ptosis and divergence manifest. (b) Primary deviation of left eye. (c) Secondary deviation of right eye with left eye compelled to fix. (d) Completed result following ptosis and muscle surgery.

FIG. 9 — Rather similar situation with unilateral ptosis and bilateral superior rectus involvement but of unequal severity. (a) Original condition. (b) Following ptosis surgery showing limitation of upper rotation bilateral. In this, one can see that there is still some residual ptosis present, with upward gaze. (c) Completion of case following muscle surgery, eyes to the front.

tient. The latter of the two patients was seen only in consultation with Dr. Francis Grant.) Surgery in the first case consisted of complete tenotomy of the levator on the left side and several weeks later a Reese orbicularis attachment to the occipitofrontalis was performed. Figure 12 illustrates a similar condition though in this instance a pseudo-Graefe syndrome of misdirected regenerating fibers was post-traumatic and a combination of inferior paralysis with levator overaction. The lid was ptosed when the eyes were directed to the front and left, and lifted in downward gaze. The surgery for both conditions is similar — it is done to convert the lid condition into a completely paralytic ptosis, separated thus from the cranial nerve which supplies the aberrant innervation, and thereafter to correct that form of ptosis by some procedure which gives maximum occipitofrontalis utilization with a minimum of the faults connected with this surgical procedure.

FIG. 10 — Asymmetric irregular ptosis. (a) Ptosis with paralysis of external and internal rectus and superior rectus. (b) Following ptosis surgery and muscle surgery. (Patient unfortunately was distressed by photographer's lighting.) (c) Ptosis on left with paralysis of internal and inferior rectus on opposite side.

FIG. 11 — Asymmetric and irregular ptosis, continued. Upper left, bilateral ptosis, torticollis, high hyperopia, horizontal, undulatoric nystagmus, ocular in type, paresis of both superior and inferior recti. Upper right, unilateral ptosis with left concomitant squint. Lower left and right, ptosis on left with paralysis of left external rectus same side, not to be confused with a protective blepharospasm.

FIG. 12 — Pseudo-Graefe syndrome. No. 1, at top, eyes front; No. 2, maximum upper rotation. Left lid lifts slightly. No. 3, downward rotation, eyeball motility limited on left. Lid also involved. No. 4, right lateral rotation. Left eye limited in right lateral rotation. Lid on left lifts strongly. Lowermost view, left lateral rotations equal in both eyes. Lid in marked ptosis.

CLASS VIII

Case 8. The patient was a colored child, 12 years of age. Duane's retraction syndrome is not too rare an anomaly. The ptosis which accompanies this condition is, in part, a mechanical form due to traction on the lid with recession of the eye ball. Recession of the internal rectus muscle, which frequently corrects the retraction syndrome, will also improve the ptosis to a marked degree. If this degree of correction is insufficient, levator advancement without the customary partial tarsectomy is adequate. In these instances, the extra-ocular muscle surgery, other than that combined with the levator surgery, is of far greater significance. The extra-ocular muscle surgery is obligatory; the levator surgery may be necessary in addition. (See Fig. 13.)

CLASS IX

Case 9. These cases are types for true plastic surgery.* Figure 14 illustrates such an instance wherein the surgery for the neurofibroma was followed later by some form of utilization of the occipitofrontalis action. It is included largely to illustrate the ideal and the indication for a Hess procedure; i.e., acquired ptosis complicated by some other plastic defects of the upper lid.

In studying these arbitrary anatomical subdivisions of congenital ptosis, one must agree that several of the classes blend with each other. Even though classes V and VI are not too dissimilar, and while together they represent approximately 10 per cent of all cases, they still are sufficiently un-alike to be listed as has been done.

The final factor to be considered in this paper is the matter of surgical correction. Surgery for the oculomotor complications follows the established rules in force for paralytic strabismus, modified only by the following: The presence of torticollis (for that indicates a struggle against diplopia); which eye is the fixing eye (for squint may be manifest because of a secondary deviation); and the danger present, in correcting a vertical deviation, of destroying all vertical elevators while attempting to eliminate this vertical diplopia.

In Fig. 8 the secondary deviation, OD, is actually greater in extent than the amount of voluntary upward rotation possible in that eye.

Levator advancements, with or without tarsectomy, demand some percentage of still intact levator action, otherwise the immediate surgical result will not remain as the permanent result. Levator surgery may be done at any

* The Virginia Medical Monthly, vol. 68, pp. 630-642, Nov., 1941.

FIG. 13 — A retraction syndrome. Uppermost left view, slight divergence only on left seen. No. 2, right lateral rotation. Marked ptosis, enophthalmos, limitation of right lateral rotation. No. 3, up and to the right. Enophthalmos on left quite evident. No. 4, upper right, down and to the right, enophthalmos even more marked. Nos. 5 and 6, rotations up and to the left and down and to the left, exophthalmos now present on the left.

FIG. 14 — Ptosis with neurofibromatosis.

time after the second year of life. Surgery of the superior rectus muscle, that is, according to the Parinaud-Motais principle, is never to be done except as a bilateral procedure. It is quite proper to operate upon both upper lids at the same time in bilateral procedures involving the levator and superior rectus muscles. This, however, should be postponed until the third year of the patient's life, if possible, simply because it is a bit easier to control, postoperatively, a three year old child. The postoperative adjustments and demands, also, in surgery of the superior rectus muscle are more exacting than are those in surgery of the levator muscles.

The three main principles to be considered in surgery for the correction of ptosis and which are all necessary at various times in these cases are: The use of the occipitofrontalis, the use of the superior recti muscles, if they are normal, and a shortening of the levator. No one operation will take care of all cases. A properly selected procedure will always give better results than any procedure selected unwisely or in error, no

matter how brilliantly that may be executed. (See Fig. 15.)

The *degree* of ptosis is not a criterion —only the *type* of ptosis present should be considered as of primary importance. The younger the patient, the more careful must one be in his surgical technique. A Frost suture should be used in all cases of children as a protection against postoperative corneal drying and damage. Infants with ptosis should have crutch glasses as soon as they are capable of sitting alone, or at least as soon as they are eight months old. The refraction for the lenses can be done always after several days of atropinization by using a few drops of vinethene anesthesia. Gross oculomotor abnormalities should be corrected to a major degree before the ptosis is corrected. There is hardly any age limit for such necessary operative procedures.

In general, ptoses classified under I, II, IV, V, and very occasionally IX, are best corrected by means of a levator advancement with usually a partial tarsectomy.

The tarsectomy is the advancement portion of a levator procedure, while that section of the levator tendon-aponeurosis which is removed is the shortening factor in the operation. The latter is always necessary, the former usually so. Ptosis classified under IX may be the exception. In class IV, dissimilar degrees of ptosis may call for differences in the amount of the tendon resection, but if a tarsectomy is to be done, this should be equal in amount in both lids. Cases in class V may not always fit into this category, and some cases in class VI may occasionally come within its limits.

If no levator action whatsoever is present in these individuals, then the surgeon must utilize the occipitofrontalis. This is especially important in younger patients. Lindner stated that with complete levator paralysis in adult life a levator advancement may be done with the knowledge that the shortening of the tendon plus the tarsectomy will be followed by a permanent cicatricial support of the formerly ptotic lid. The author concurs with him in this observation.

Ptosis classified under III is the ideal indication for the Motais-Parinaud technique. There are several satisfactory modifications of this technique; that which the author likes best is the Shoemaker modification. Ptosis classified under VI may occasionally fit into the limits indicated for this operative technique.

For ptosis classified under VI, and in all children with completely paralyzed levators whose cases are classified under I, II and IV, the correction should be done either by a Gifford modification of the Machek operation, a Hunt-Tansley procedure or by the Reese technique which corrects more adequately at the canthal angles. After late childhood, the patient can always undergo resection of these occipitofrontalis cicatricial tracts and then any other procedure desired can be carried out, if indications at that time have changed.

For ptosis classified under VI early extra-ocular muscle surgery is of great importance and should be completed satisfactorily before the ptosis itself is corrected. If not, one may find two difficulties arising—first, a struggle against the re-establishment of diplopia, and, second, some return of the formerly corrected ptosis, as a defense mechanism.

For ptosis classified under VII and VIII a complete tenotomy with tenectomy (resection but without re-attachment) of the levator should be done, followed several weeks later by a Reese orbicularis fibers transplant to the occipitofrontalis. In doing this tenotomy, the surgeon must be careful that he also cuts free the lateral horns of the leva-

tor where they are attached to the canthal ligaments. For ptosis classified under VII this same operation may at times be needed. For ptosis classified under VIII, the same is true, if the ptosis which remains after the resection of the fibrosed extra-ocular muscle suggests that fibrosis is also present in the levator muscle and its aponeurosis.

Ptosis classified under IX is a true plastic defect of the lid and should be corrected in the major number of instances by a Hess procedure. It may be possible, at times, to find and re-attach the levator, however. If so, the surgeon may find that he must transplant a piece of cartilage from the ear into the lid to replace the lost tarsus, otherwise lid notching will develop during the wide elevation of the upper lid. It is a fact, the Hess procedure seems to have its best indication, not in congenital ptosis, but in the acquired ptosis of other accompanying lid defects, as in a reconstructed lid following a major traumatism to that lid.

RECAPITULATION OF SURGERY

From the standpoint of available technique for the correction of congenital ptosis versus acquired ptosis, and excluding that surgery necessary for the various oculomotor complications, the following procedures are recapitulated as indicated:

In utilizing the occipitofrontalis one must consider the Gifford modification of the Machek operation, the Hunt-Tansley operation and the Reese orbicularis transplant. The Gifford modification is indicated in the cases of slightly older children with complete ptosis wherein epicanthus is outstanding; the Reese orbicularis transplant is indicated in cases in which the jaw-winking reflex is present; the Hunt-Tansley operation for all other cases within this subdivision of indications. The various other forms of this basic technique such as Lexner's principle and the Hess introduction of Pagenstecher sutures, the Panas operation, the direct use of fibers from the occipitofrontalis (Robert's technique for the Freeland Fergus operation), Dransart's use of absorbable material, including fascia lata according to Derby, Wiener and even Dickey's operation, are all proper procedures but their indications lie in the field of acquired ptosis, especially ptosis associated with other plastic defects of the lids and of the tissues immediately adjacent to the lids.

FIG. 15 — Unilateral Motais operation. Surgery done ten years prior to time middle photograph was taken. Photophobia, exposure keratitis with lagophthalmos, recurrent corneal ulcers, vision: hand movements only. Operation not only unilateral but also done upon a paralytic (congenital) superior rectus. See middle photograph for upward rotations. Lower photograph, the lagophthalmos present with maximum closure of the lids. Surgery to correct. Resection of the lid and the superior rectus adhesions followed by a levator advancement, this completed very recently and successfully. Upper photograph taken following reoperation.

The indications for and the permissibility of the utilization of the superior recti muscles has, as stated before, a narrow field. The operation is only for bilateral conditions, if both eyes are still present and are functioning. (See Fig. 15 for the unwise use of this technique.) The modifications of this operation for congenital ptosis which the author considers as outstanding are either Kirby's or Shoemaker's, which are to be performed bilaterally, at the same operation. Young's operation, although presented later, is essentially the same as Shoemaker's. Dickey's superior rectus fascia lata transplant is not recommended for unilateral congenital ptosis. This should be reserved for bilateral ptosis. Fascia slings as such, however, while hardly necessary for congenital ptosis, unless the case is an old neglected one, are a most valuable procedure when used properly. The technique is ideal for certain complicated situations. It has been used by the author in cases of acquired ptosis for the elevation of reconstructed lids (both flap and free skin graft corrections) with satisfactory results, and in two instances for re-operation in the cases of older patients wherein earlier surgery had unfortunately included the resection of considerable skin surface of the lids. (See Fig. 16.)

FIG. 16 — Old incomplete correction for ptosis. Patient late adult life. Lower view, same patient immediately following levator sling surgery.

The firm attachment which this operation gives lends itself well to the heavy upper lids in such cases. The Wheeler levator-superior rectus operation should apply to this same class of cases. Further, these two operations might well be considered as re-operative techniques in instances wherein earlier surgery had either failed or was insufficient in correction, considering always that the superior recti muscles are normal in function. The Trainor operation, also a levator-superior rectus muscle procedure, has only simplicity and rapidity of technique to offer, with other more important factors against it. The disadvantages are that here again is a superior rectus procedure improperly recommended as a unilateral operation, which, if successful as a ptosis operation, means a potential hypophoria and a surgical failure in the presence of that so common superior rectus-levator joint paralysis. The superior fornix adhesions formed are an even greater deterrent to its use. They prevent normal lid eversion, and are a constant drag upon the lid and the eye ball with lateral rotations and with convergence. The operative adhesions formed are inelastic and rigid. Its two virtues might indicate its use under not too uncommon circumstances, such as the permanent ptosis of cerebrovascular situations in older patients.

Levator advancements, with or without tarsus resections, have the greatest possibilities in properly selected cases of congenital ptosis. Inversely, their usage in cases of acquired ptosis is relatively unimportant, except for two conditions: (1) The ptosis which occurs following the enucleation of an eye. (See Fig. 17. This figure illustrates a satisfactory correction by the external route resection, the Everbush operation.) (2) That following partial traumatic section of the levator. (See Fig. 18.) This external route operation probably applies best to unilateral acquired ptosis, wherein some levator action is still residual, and when

FIG. 17 — Ptosis from an old enucleation. Case completed with a bone ball implant into Tenon capsule, and free graft reconstruction of the inferior cul-de-sac prior to the Everbush ptosis operation.

FIG. 18 (a) — Incomplete ptosis following partial traumatic section of left levator. (b) Following re-attachment of the levator.

FIG. 19 — (a) Lagophthalmos following Hess ptosis operation for unilateral ptosis. (b) Return of ptosis following removal of the cicatrices formed by the Hess procedure. (c) Case immediately after external route levator advancement. Sutures and pearl buttons still in place. Some edema of upper lid still present. Very satisfactory lid fold. Lagophthalmos no longer present.

the condition is also complicated by other plastic defects not involving the loss of soft tissue or by cicatricial contractions, such as neurofibromatosis, at times, bony defects of the orbital wall, retrobulbar neoplasms for which orbitotomy has been successfully performed, the ptosis of chronic trachoma and after symplepharon corrections. Also, it can be used after reoperations wherein a Hess procedure had to be undone, to permit a more satisfactory cosmetic result. (See Fig. 19.) It is understandable that the cause of some types of acquired ptosis cannot be corrected by any surgical procedures whatsoever. Myasthenia gravis (Fig. 20) and chronic progressive external ophthalmoplegia (Fig. 21) are the two best examples of this class of cases. Many other forms of acquired ptosis, by reason of their etiology, result in an almost complete to complete levator paralysis, hence exclude levator advancements except in individuals well along into adult life.

FIG. 20 — Unilateral ptosis due to myasthenia gravis. Eyes opened and closed. Condition arrested. Patient returned to work following prostigmine therapy.

FIG. 21 — Ptosis bilateral, more marked on left. Chronic progressive external ophthalmoplegia. Lower photograph — patient wearing crutch glasses.

CONCLUSIONS

A classification of the types of congenital ptosis is presented which has as its criteria unilaterality or bilaterality of the defect and the presence or absence of other extra-ocular muscle abnormalities, as they appear in different groupings. The surgery indicated for ptosis classified in the various subdivisions is discussed to emphasize the necessity for varied procedures to meet adequately the varied indications which present themselves. The improper and unwise use of various procedures is also emphasized. Most of these surgical procedures have sound anatomical principles underlying their use, but only when they are used to combat specific situations. Complete failures, even partial failures, are unfortunate because these are unnecessary. However, new complications due to surgery carried out are worse. A recent statement of Ruedemann's must continue as our creed: "All surgical procedures in ophthalmology should be selected for the best surgical end results for the patient."

This paper is closed with a plea for earlier diagnosis and for treatment as early as is possible in all individual instances of blepharoptosis. Further, the variable aspects of congenital ptosis cannot be stressed too emphatically. A statement made in 1940 by Dunnington in regard to the confusion connected with ptosis surgery seems to be ideal for closing. "If careful attention is paid to the findings at the preoperative exam-

ination and if the surgeon is willing to employ the procedure especially suitable to each patient rather than to rely on the one operation which he prefers to perform, the results will be more uniform and a more accurate prognosis possible."

DISCUSSION

BERNARD J. LARKIN, M.D., Indianapolis: Doctor Spaeth's able presentation has covered the subject so well that there is nothing to add. The fine organization of his material makes it easy for us to follow him to his conclusions. He has properly placed congenital ptosis, and this is certainly sufficient reason for our appreciative consideration of his research and study.

He has stressed the importance of early diagnosis and treatment, for 37 per cent of the cases in his series were complicated by factors other than epicanthus and ptosis. He suggests that the use of the crutch glasses after atropine refraction will assist in overcoming these complications and thereby operation may be postponed until the child is four or five years old; that not infrequently paralysis of the superior rectus is of equal importance in causing other complications of ptosis which are more serious than the cosmetic blemish; that the anatomical classification decides the surgical procedure, and he emphasizes the fact that one operation will not correct all cases of ptosis.

His classification has nine subdivisions. The first impression may suggest that several of these classes are similar, but after careful analysis one must agree with Doctor Spaeth that sufficient conditions exist to differentiate these subdivisions. Moreover, this becomes especially significant in considering the analysis and presentation of the cases illustrative of the subdivisions. In referring to these, let us recall class I—unilateral ptosis without involvement of the superior recti muscles; class V — unilateral ptosis with weakness of both superior recti muscles, more marked, however, in the homolateral eye, and class VIII — ptosis with the Duane's retraction syndrome. For these types of cases shortening of the levator with partial tarsectomy would be the ideal procedure to follow. In class VIII recession of the internal rectus muscle may correct this retraction syndrom as well as the ptosis; however, if this is not sufficient, an advancement of the levator without tarsectomy will probably correct it. Other surgery was necessary in case V in which there was a recession of the overacting right inferior oblique, recession of the left inferior rectus and advancement of the left superior rectus muscle. This illustrates the advisability of early extra-ocular muscle surgery before the ptosis is corrected.

"The utilization of the superior recti muscles has a more narrow field of indications and permissibility." Doctor Spaeth seems to be the first to advocate a still further narrowing of the utilization of the superior recti muscles in a bilateral condition by limiting the Motais operation with a Shoemaker or Kirby modification. In a former paper he set forth his reasons for this opinion. The postoperative care of patients undergoing superior rectus muscle surgery is also more demanding and exacting than of those undergoing levator muscle surgery.

Utilization of the occipital frontalis, when really indicated, is a good procedure to follow, as the author has shown. In class II, unilateral ptosis with involvement of the homolateral superior rectus muscle, and in class IV, bilateral ptosis with involvement of the superior rectus muscles, the Tansley-Hunt procedure was chosen, while in class VI, ptosis with more or less complete third nerve and even sixth nerve paralysis, the Reese procedure is indicated. In class VII, ptosis with the classical jaw-winking reflex, a levator tenotomy was first performed, and later a Reese orbicularis attachment to the occipitofrontalis was carried out.

The exaggerated epicanthus which occurs in those children with bilateral ptosis should not be operated upon until the child is older, as the condition frequently adjusts itself when the adult facial growth is reached. However, in exceptional cases the Gifford technic is the method of procedure.

I have touched upon only those points in treatment of congenital ptosis that seem to me to be most striking and pertinent.

In conclusion, I should like to repeat Doctor Spaeth's closing words, "A plea for earlier diagnosis and as early treatment as is possible in individual instances."

EDMUND B. SPAETH, M.D., Philadelphia: Thank you, Doctor Larkin, for your discussion.

That portion of the paper which I could not read because of time limitations contains a detailed discussion, and very frankly in de-

tail, about how I feel regarding various surgical indications and their application. For instance, with the exception of a complete ptosis in children, I do not feel that the occipitofrontalis procedure is permissible except in cicatricial ptosis of adults or cicatricial ptosis of children if there is complete levator paralysis.

I think the jaw-winking reflex has been rather badly handled by most of the neurosurgeons. It is unfortunate that those cases get to the neurosurgeons, as these patients end up as neurosurgical problems for the rest of their lives. It is such a simple procedure to convert this type of ptosis into a completely paralyzed ptosis and then carry on from there with the occipitofrontalis utilization by means of Reese's use of fibers and cicatricial bands from the orbicularis to the occipitofrontalis. Thank you.

In Memoriam

Louis Joseph Sebert.....Toronto, Canada
December 2, 1942

Frank E. Detling.....Los Angeles, California
December 25, 1942

Robert Lapsley.....Keokuk, Iowa
January 5, 1943

Frank A. Hartley.....Springfield, Ohio
January 18, 1943

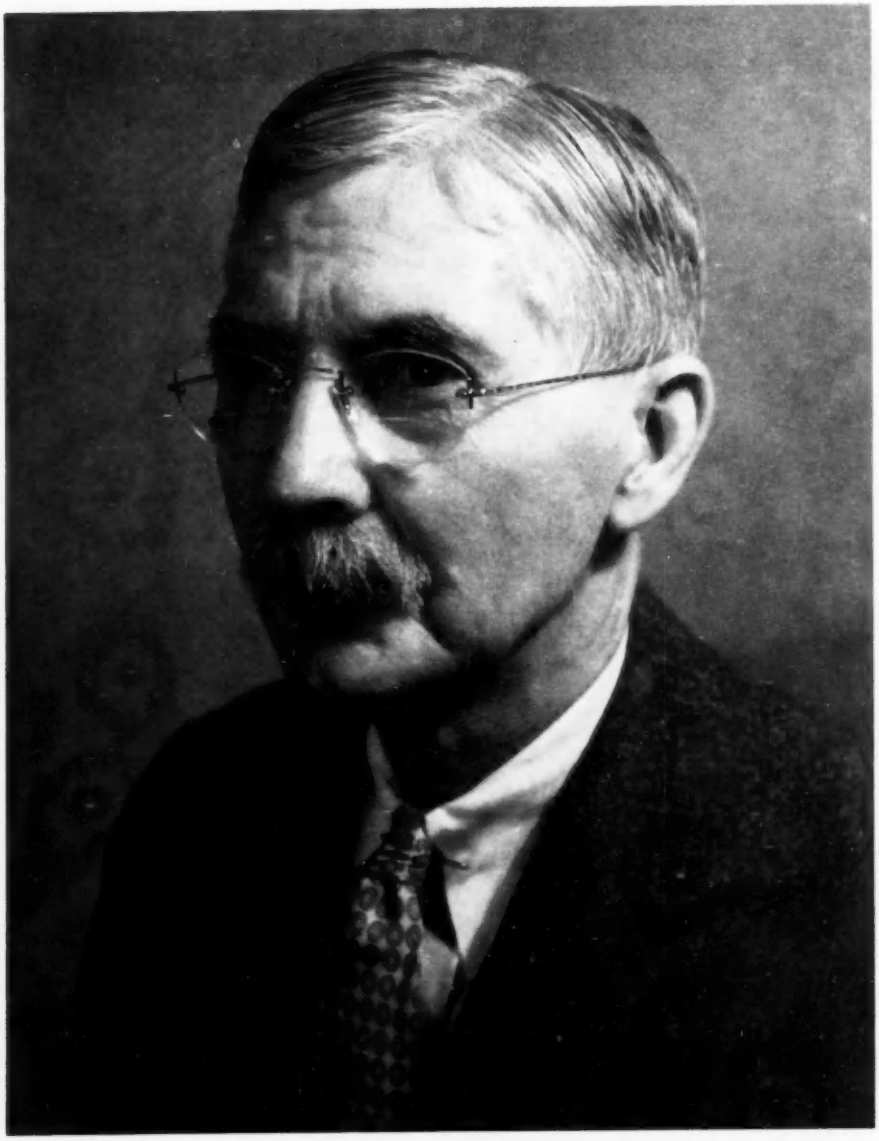
Willard G. Mengel.....Camden, New Jersey
January 27, 1943

Carl B. Wagner.....Chicago, Illinois
February 1, 1943

John F. Barnhill.....Miami Beach, Florida
March 10, 1943

Walter J. Wherry.....Omaha, Nebraska
April 1, 1943

In Memoriam



JOHN FINCH BARNHILL, M.D.

1865-1943

JOHN FINCH BARNHILL, M.D.

1865-1943

Dr. Barnhill was born on January 2, 1865, on a farm near Nora, Indiana. He received his early grammar school and high school education in Westfield, Indiana. He was married to Celeste Terrell who survives him. They had no children.

Dr. Barnhill began his career as a school teacher. He later entered the Central College of Physicians and Surgeons, which eventually became one of the component parts of Indiana University School of Medicine as it is today. He was graduated in 1888.

After practicing general medicine a short time in Indianapolis he became interested in otolaryngology. He did not, however, simply announce that this was the special field of work he had chosen and begin practicing it without any preparation, as many men did at that time. He sought medical centers where he could receive this training. In our own country he found this instruction in Chicago, New York City and Philadelphia; and abroad he found it in London, Paris, Berlin and Vienna.

Dr. Barnhill was president of his local medical society in 1924, of the American Laryngological, Rhinological and Otological Society in 1928, the American Academy of Ophthalmology and Otolaryngology in 1931, the American Laryngological Association in 1937, and was Chairman of the Section of Rhinology, Otology and Laryngology of the American Medical Association in 1903-1904. He was a Fellow of the American College of Surgeons.

Dr. Barnhill took a very keen interest in general medical affairs. He was always interested in the welfare of the younger men who were working in his chosen specialty, and he never failed to promote their welfare if they were deserving of such promotion.

His interest in medical education dated from the time he became an assistant in the Department of Otolaryngology in Indiana University School of Medicine and continued to his demise. Dr. Barnhill was chairman of this department; he was chairman of the Department of Head and Neck Surgery, and he developed an anatomical and clinical course in otorhinolaryngology. Out of this course came his work on the anatomy of the head and neck which was unique in that he presented it in monologue style. One would have to be a student and co-worker of his to fully appreciate this work.

Dr. Barnhill, with Dr. Ernest deW. Wales, published a work on otolaryngology. He read numerous papers before the special societies, many of which were later published. He never feared to express his own opinions in a discussion. In the literary fields his hobby was along historical lines, and one of his outstanding productions was "Hatching the American Eagle." He was granted the degree of Doctor of Laws by his alma mater, Indiana University, in 1929.

He was one of the founders of the present Indiana University School of Medicine. He was responsible for obtaining the endowment for the Robert Long Hospital which was the first teaching hospital of the medical school center.

All the members of his staff in the University liked to address him as "Uncle Jeff" and he was quite fond of this bit of familiarity when so addressed. He enjoyed a good story and liked to tell one. During his last years he was thrilled when he could be present at the "Barnhill" dinners given for him at the Academy meetings by the fellows who had taken his anatomical and clinical courses.

Dr. Barnhill was physically handicapped throughout his life by being afflicted with asthma. During the last ten years polyarthritis was added, from which he suffered intense pain. Despite these afflictions he lived to the ripe age of 78 years. He died on March 10, 1943, at Miami Beach, Florida, where he had made his home for the past ten years.

CARL H. MCCASKEY

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Editorial

The Convention

Plans are now well advanced for the forty-eighth annual meeting of the Academy, which will be held at the Palmer House in Chicago October 10-13, 1943.

In harmony with conditions brought about by our government's requirements, the entire program is being constructed to provide the greatest amount of instruction in war medicine that can be concentrated into one streamlined activity. Approximately 300 hours of didactic instruction in ophthalmology, otolaryngology and plastic surgery of the head and neck will be given by America's foremost teachers. The instruction courses will provide a variety of subjects covering refraction, pathology, diagnosis and treatment of disturbances of motility of the eyes and will include lectures and demonstrations on matters pertaining to military requirements. Recent advances in all phases of otolaryngology, including the results of recently completed research in hearing, will be demonstrated by lectures and by motion pictures made expressly for the Academy by the Committee on the Conservation of Hearing. The number of courses available to any member has been increased by 50 per cent by scheduling three periods of instruction each morning instead of two periods as in former years. In order to meet the growing expense of the convention, the Council has found it necessary to raise the fee for instruction courses to $1.50 for each period.

The convention will open on Sunday afternoon, October 10, with a joint program at which Dr. Joseph E. Raycroft, Chairman of the Committee on Student Health of the American Medical Association, will be the guest speaker. Following the scientific program, the President's reception, the most popular hour of the meeting, will be held in the Red Lacquer Room, where old acquaintanceships will be renewed and the new members welcomed.

On Sunday evening there will be two attractive symposiums. The Committee on the Conservation of Hearing will present a series of papers by distinguished teachers on a subject of vital interest to all otologists. The Committee on Industrial Ophthalmology will present a program of unusual interest.

These functions are practical in scope and are added features of the stepped-up Academy service to the Nation.

There is every indication that the attendance will equal or surpass that of last year, and the plans of the Program Committee have been made on this presumption. Members will do well to take into account the necessity of early arrival in Chicago and make hotel reservations well in advance of the meeting. Please register early, secure your course tickets early and don't miss a minute of the meeting.

The Directory

The Academy roster now contains 3,368 names and addresses and comprises a volume of 70 pages. The alphabetical list is followed by a geographical list for convenience in reference. Every member of the Academy wants to have on his desk this list of the outstanding ophthalmologists and otolaryngologists. In order to make the roster easily available and to avoid the necessity of keeping one of the regular issues of the TRANSACTIONS from its proper file, the roster is supplied to all members as a supplement of the TRANSACTIONS, bound in its own cover for daily desk use. As the cost of setting up the directory is considerable, only a few extra copies will be made for replacement, and for these will be charged a fee of one dollar to cover the cost of printing and mailing. The complete roster will be included in the bound volume of the TRANSACTIONS.

Specialist's Role in the Armed Forces

Determination of the role to be played by the certificated specialist in our armed forces takes on more than an academic interest now that we are in the second year of war. The rapidly enlarged medical corps of the army and navy to give service to the men in the expanded army and navy is only one reason for our interest.

The American Academy of Ophthalmology and Otolaryngology has done much to translate into a factual procedure the certification of specialists in the branches of medicine which are our concern. This was done to furnish better specialists' service to the public. By the same token we cannot strip ourselves of the obvious corollary to all this useful activity by avoiding deep concern for the status of our specialties in the armed forces.

In the regular medical establishment, the extent of specialization is limited. The regular medical officer is forced to a level of partial specialization, while maintaining a broader contact-base with general medicine.

The New York Committee on Mental Hygiene has published some interesting figures recently. Dr. Lawrence S. Kubie takes the regular medical establishment of 1938 as a "yardstick." At that time there were 1,042 medical officers on duty with the army. There were 815 regular medical officers in the navy. Assuming the medical population of the country as comprising 180,000 physicians, there were then about 1,800 regular medical officers on duty. This gives a figure of one per cent of the medical population in the regular medical establishments of the army and navy.

Kubie reports that the membership of the American Academy of Ophthalmology and Otolaryngology was approximately 2,600 in 1938. Among our members there were then only 22 regular medical officers of whom 8 came from the army and 14 from the navy. The criteria of representation should have approximated 26, or one per cent. There were no army or navy medical officers among the members of either the Triological Society, the American Otological Society, the American Laryngological Society or the American Broncho-

Esophagological Association. As for the number of diplomates of the American qualifying boards, it is reported that from among the 1,300 ophthalmologists who were diplomates in 1938, there were 16 medical officers which is an increase of three over the one per cent—the standard figure. From among the otolaryngologists who were diplomates, there were 35 medical officers as compared with 2,770 non-military diplomates. Here, too, there was an excess of seven over the one per cent standard figure.

In 1939, approximately eight per cent of the physicians in the United States were diplomates of some National board.

This is the peace-time picture of the part our specialties are playing in the armed forces.

With the rapid integration of many of our members in the service, it would be timely to inquire how they are being utilized. We should endeavor to ascertain the value to them of their qualifying certificates while serving in the armed forces. If they are properly assigned to the tasks for which their education and experience has fitted them, then the American boards can be proud of their achievements. We should know that the millions of our youth within the armed forces have in medical attendance the "cream of the crop."

We are aware of carefully prepared reports on the medical potentialities of the groups concerned with our specialties. We should use the time-honored medical method of "follow-up" to determine how the eye, ear, and the nose and the throat services of our military hospitals are functioning.

The Future

There are many of us who have devoted a large part of our professional life not only in treating the sick and in the research fields which widen the horizon of medical knowledge, but also in the development of the American system of practicing medicine in a manner the results of which are found in successive reports of better morbidity and mortality figures.

There are trends in the public thinking of which we must take note. Plans are contemplated and in the making which make the hospital, rather than the doctor, the center of practice. If this idea spreads, the practice of specialization will sink to a general level of mediocrity, and medicine as we know it, in general, and in our specialties, as branches of highly technical modalities, will go into eclipse for some decades.

This is the time to plan. This is the time to study the trends in public affairs to preserve *in* the "American Way of Life" one of its greatest contributions to human welfare.

No plan should be encouraged which destroys what has laboriously been achieved during the last two decades of special practice.

Letter to the Editor

Feb. 5, 1943
Lt. Col. Derrick T. Vail
Office of Chief Surgeon
A. P. O. 871, New York

Dear Bill:

I have just returned from a swing around the plastic surgery centers in the Islands, except for seeing Gillies. The lid repair is very interesting and first class. No Wheeler implants are used in ectropion except very small ones. Usually, 1st, excision of scar and, 2nd, very large Thiersch grafts from inner side of upper arm (3-4 times the area to be covered) are placed and held by a stent and a few sutures. The rough edges are trimmed. Usually upper and lower lids are not replaced at the same sitting, although this varies. Later, the ridges of excess skin are excised. Eyebrows are implants from behind the ear. Epitheli-

um is used to line the lids, but it isn't very satisfactory. Great care is taken to anchor the external canthus to the periosteum, and of course the internal as well. There is some neglect to approximate lid lacerations at the first instance, and I have been emphasizing this point in my teaching to the field officers. I am very much impressed with the good results obtained. The lid grafting seems much better in giving natural wrinkles and folds than the Wheeler method, and there is a great advantage in the use of so few sutures. Sliding flaps are rarely used, except as protection in the first instance, when the eyeball is exposed on the field. Some thought should be devoted to this point. Depressed eyeballs are elevated sometimes with cartilage wedges subperiosteally, but it is not so hot. The rest of the plastic work on hands, face, etc., is most impressive.

Yours,

DERRICK

Letter to the Editor

Gorgas Hospital
Panama Canal Zone

Dear Doctor Benedict:

Gorgas Hospital is the largest and best equipped hospital in the Zone. While it is operated by the Panama Canal Government, it is chiefly staffed by Army medical officers. The hospital is open to all white and colored employees in the Zone, as well as all members of the armed forces of the United States.

Our Eye and Ear Clinic is divided into the Eye and Ear, Nose and Throat Sections and is staffed with five physicians. There are well over 5,000 patient visits per month, and eight to 10 operations daily five days a week. The eye conditions do not differ materially from those which one might see in any active clinic in the United States, except in regard to the proportions of the different ocular conditions. Traumatic conditions of the eyes are especially common among the Negroes, Central American natives and soldiers. Almost every day we see five or six cases of foreign body on the cornea. Subluxated lenses, detached retinae and cataracts are not uncommon. At least two or three new cases of pterygium are seen daily, especially in the Jamaicans and Central Americans and not infrequently in the soldier who has been in the tropics for some time. They seem to be of a particularly virulent character, and unless carefully handled, recur with alarming proportions. Whether the pterygium be transplanted or excised, we have records to show the good and bad side of each of these procedures, and certainly no preference can be clearly stated as yet.

Chronic simple glaucoma in the youthful Negro (native of Jamaica, Barbados or Martinique) is not uncommon and appears especially resistent to eserine or pilocarpine. Repeated clinic attendance and the faithful use of drops by these patients is not satisfactory so that we have felt justified in offering surgery (cyclodialysis or corneoscleral trephine) at an early date.

The various forms of uveal tract involvement are strikingly common. Syphilis, tuberculosis and leprosy appear to be the most common etiologic agents in the Negro and native, while tuberculosis and some form of focal sepsis seem to be the chief etiological factor in the soldier. In regard to the iritis seen so frequently among the lepers of our fair-sized colony, I have been struck by the ineffectualness of atropine when used topically in the leprotic eyes even before any dense adhesions have taken place. However, the subconjunctival injection of equal parts of two per cent atropine, five per cent cocaine and 1-1000 adrenalin has been surprisingly effective. This measure, coupled with injection of milk intramuscularly has alleviated about 90 per cent of the leprous iritis cases within a few weeks when encoun-

tered early. This may sound like an unimportant statement but anyone who has been confronted with iritis in leprosy is well aware of the alarming irreversible complications that can occur in these patients' eyes if not treated satisfactorily.

In the clinic, cases of senile cataract appear in about the same percentage of elderly patients as is seen in North American clinics, but traumatic cataracts are quite frequent.

Syphilis, with its various ocular complications, forms an unending procession of pathology. We have been curious about the infrequency with which we are seeing strabismus cases among the Negroes and natives. It is by far more common amongst our American soldiers if we are to form any opinion from a cross section of our practice.

We are constantly on the alert for the various fly infestations in the form of larvae about the skin of the eyelids and conjunctiva. Ocular myiasis, while not common, has been of intense curiosity to me.

Several of the trees and plants about the outposts contain a highly irritative sap which when encountered even in the slightest dilution causes a most intense keratoconjunctivitis. One is known as the Manzanilla plant, popularly called the Beach Apple. While no permanent damage appears to follow the reaction from these irritants, they often cause incapacitation for a week or 10 days through ocular distress alone.

Fully one-half of my patients speak and understand nothing but the Spanish language. I have no way of testing the extent of their Spanish vocabulary, but I am acutely aware of my limited knowledge of this language. The following is a brief recitation of what may and can happen to a North American ophthalmologist practicing his art amongst his Latin American neighbors: A dear 64-year old Colombian had been under our care for a low-grade chronic uveal inflammation. He was given two prescriptions . . . one calling for 20 drops of potassium iodide in milk twice a day and the other one instructing him to use one drop of two per cent atropine sulphate in his affected eye twice daily. Within 24 hours, he was brought back with acute atropine poisoning. He had taken the atropine by mouth and put the K. I. his eye. Fortunuately, he has survived even this reversed therapy and is now one of my favorite patients.

Sincerely yours,

R. D. HARLEY
Capt., M.C., U. S. Army

Announcements

Industrial Ophthalmology

The Council of the American Academy of Ophthalmology and Otolaryngology, as a part of the program for the October meeting, is making plans for a special meeting devoted entirely to industrial ophthalmology. This is an open meeting and will be held Sunday evening, October 10.

It is proposed that a symposium be presented on several subjects which seem to be of general interest to those ophthalmologists who may be practicing in this field.

As a tentative program it is planned to have four or five speakers — two laymen and three ophthalmologists. One paper will be given by a safety engineer, another by a person interested in employment of personnel and three others by ophthalmologists. For these papers, the suggested topics are: "First Aid to Eye Injuries," "The Management of Intra-ocular Foreign Bodies," "The Use of Chemotherapy in Eye Injuries," "Program of Ophthalmic Service for Small Industrial Plants." A period for questions is planned as a part of this program.

ALBERT C. SNELL, *Chairman*

Home Study Courses

The 1943-44 Home Study Courses will be started *September 1,* but registrations will close on *August 15.* Nine months will be required for completion of the courses. Applications are now being accepted by the Executive Secretary-Treasurer, when accompanied by the registration fee of ten dollars. Application blank may be obtained from the office of the Executive Secretary-Treasurer, 239 One Hundred First Avenue Building, Rochester, Minnesota.

The courses will be conducted essentially as in the past. On the first of each month, the registrant will be sent a selected list of reading. At the end of the month, he will receive a set of 20 questions, covering the reading of the past month. These questions are to be answered within 14 days and then returned to the Committee. The answers will be corrected and the papers graded by members of the faculty and the papers returned to the registrants for permanent inclusion in the ring folder that will be furnished at the beginning of the course. Undue delay in the return of the examination paper by the registrant will result in the paper not being corrected. Registrants will please refrain from writing to the Committee asking why their corrected examination papers have not yet been returned. The Committee and the faculty will do their utmost to get the papers back to the registrants as soon as is possible.

Owing to the limitation of the number of faculty members who perform the laborious task of correcting examination papers, it was necessary this past year to limit the number of registrants submitting examination papers to 50 in ophthalmology and 50 in otolaryngology. In this, preference was given to bona-fide residents as will also be the case for the coming year. All other registrants of the Home Study Courses will receive monthly the reading lists and the examination questions. The answered questions must not be submitted to the Committee for the papers cannot be corrected. Instead these registrants will receive a model set of answers to the examination questions, se-

lected from the best answers submitted. Judging from the responses of this past year, this procedure has met with satisfaction.

Inasmuch as these courses are offered by the Academy solely for the benefit of members and prospective members, no certificate will be given upon completion. But upon request, the Committee will send the registrant a letter stating that he has completed the Home Study Course in ophthalmology or otolaryngology.

Registrations will be accepted for either ophthalmology or otolaryngology, but not for both, from any one registrant in any given year.

THE COMMITTEE

Ophthalmology
Harry S. Gradle, M.D.
Daniel Snydacker, M.D.

Otolaryngology
Frank J. Novak, Jr., M.D.
L. Benno Bernheimer, M.D.

The William P. Wherry Memorial

The Council authorized the establishment of a Memorial to our late Secretary-Treasurer in the form of a paid-up Life membership in the Academy to the person selected annually by the American Board of Otolaryngology as the most outstanding candidate examined. This was to be financed by the interest on $16,000.00 to be raised by voluntary subscription and invested in War Bonds. (The interest on that amount is $320.00 per year, $20.00 of which would be applied to the initiation fee and $10.00 to the annual dues for 30 years.) This form of Memorial was considered most appropriate because (1) It provides the type of opportunity to outstanding candidates for membership in the Academy that lay closest to Bill's heart; (2) It stimulates the sale of War Bonds, and (3) It provides members the opportunity of showing the respect and admiration for Doctor Wherry in a manner that accrues to their own interest.

Thus far less than one-half of the objective has been subscribed, and a deficit remains that threatens to vitiate a splendidly planned Memorial Tribute. If you haven't contributed yet, send your check right away to the Executive Secretary-Treasurer, ear-marked for the Wherry Memorial. If you prefer, send in a War Bond or two or three that you have purchased. All these contributions are deductible from your income tax.

Conservation of Hearing

The Committee on Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology is sponsoring a program for the diagnosis and treatment of hearing defects in school children. It is their desire to formulate a practical plan which can be adopted by the various states or by local communities.

That there is need for such a program is evidenced by figures indicating that of the 50,000,000 school children in the United States, at least 60,000 are totally deaf, 3,000,000 are hard of hearing and 4,000,000 have speech defects attributable to impaired hearing. It is said that in each school room from one to three children have defective hearing. Five per cent, or 2,500,000, school children in the United States have some degree of impaired hearing.

Some work has been done in the detection of defective hearing in school children. Except in a few instances this has been on a small scale with little done in the detection or treatment of earlier types of hearing impairment.

Incomplete surveys in Iowa indicate that not much has been done as far as remedial measures are concerned, with

the exception of the excellent work being done by the Iowa State School for the Deaf.

The desire of the Committee, as stated above, is to suggest a plan which may be adopted by the various states with the following purposes:

1. To establish routine testing of all school children at once and thereafter in the third, sixth and ninth grades.
2. To bring all cases with defective hearing to the attention of the physician for treatment when indicated. Sufficient emphasis has not been placed on remedial measures to arrest the progress of deafness.
3. To provide special education for those with hearing losses which are not sufficiently severe to necessitate institutional training.
4. To detect all cases with hearing losses severe enough to require training in the school for the deaf, which provides vocational training, rehabilitation and eventually employment for children as they grow into adulthood.

Certain publicity should take place before submitting a bill to the legislatures of the various states. The problem should be presented to the various county and state medical societies, service clubs, American Legion, parent-teacher associations, school officials and the public in general.

Following this, a bill should be prepared by the legislative committee of the state medical society with the cooperation of the Department of Public Instruction and the Board of Education. The bill should contain specific information in regard to the funds necessary for salaries, equipment and travel expense. Economy should be advocated. The bill should, in our opinion, appropriate funds for testing of children in public schools, and most important, provide for referral to private physicians and clinics those cases which may benefit from treatment. Cases with more marked hearing losses should be referred to an advisory committee for recommendations in regard to education and rehabilitation.

For a state with a population of 300,000 school children, the following organization is suggested:

The personnel to consist of a director, five technicians, secretary and state agent; an advisory committee consisting of the director, state agent, superintendent of the State School for the Deaf, at least two otologists, instructor of speech, director of Public Health and two lay members. The duties of this committee would be to review and make suggestions for those cases referred for educational rehabilitation.

The testing should include all children in the third, sixth and ninth grades as well as other special cases brought to the attention of the director. The tests should be given, as a minimum in these grades, at least every three years in each local school unit and more often if possible.

The method of operation might be as follows: A schedule could be formulated by the director. The superintendent of the local school unit would be advised at least a month in advance of the time of the tests. The local county medical society would appoint an otologist to conduct the physical examination. The school officials and school nurse would assist in the examination. At the appointed time the audiometric technician would arrive and select a suitable place for the examination. Following the examination the cases with definite hearing impairment would be referred to the examining otologist. The parents would accompany the child in order to give an adequate history and to receive an explanation of the examination. Those cases which are considered reme-

dial would be referred to the general practitioner who in turn would refer the patient to an otorhinolaryngologist. Indigent cases would be referred to the various clinics. Cases with more marked hearing losses would be referred to the advisory committee who would recommend private or public schools for special instructions.

It would be the duty of the state agent or social worker to keep a careful follow-up of all cases with impaired hearing.

No attempt has been made to furnish specific information in regard to the necessary funds, technique of testing, et cetera, as this is being compiled at the present time and will be presented later.

DEAN M. LIERLE, M.D.
Chairman, Committee on Conservation of Hearing

Industrial Ophthalmology

While the Joint Committee has been functioning informally rather than meeting formally under pressure of the increased load of war work, it has, nevertheless, not been idle. In continuing the research work on Industrial Visual Problems, the effort has been to delineate a basic and exceedingly flexible program, which, when presented to any industry can be streamlined to the purposes of that specific industry, making it possible at the same time for that industry to set its problems against the over-all picture.

The Committee has acted also as a clearing house for queries that come to the American Medical Association, to the National Society for the Prevention of Blindness (two members of the Joint Committee serve on advisory committees of the N.S.P.B.), as well as innumerable queries from individuals and individual industries. These queries have varied from questions relative to new chemicals and their effect on the eyes to testing methods, therapeutic measures and medico-legal matters. All have been checked and answered by some member of the Joint Committee if full information were available. Some times the answers were based on further investigation.

The Committee issued a warning bulletin on epidemic keratoconjunctivitis in conjunction with the United States Public Health Service which was distributed by the Industrial Health Section of the A.M.A. at their request. It has also worked very actively in assisting county and state medical societies in preparing warning bulletins in individual danger spots such as the middle west.

At present, the Committee is in the process of arranging for research on radiation, the amount, intensity and time element of infra-red and ultraviolet in new industrial processes and their possible effect, if any, different from known radiation factors. This work, when completed, will we hope be an important contribution both to safeguarding hazardous jobs adequately and to clarifying the medico-legal picture.

The Committee through one of its members has been very close to some of the shipyard eye problems, particularly those of "flashes," and further developments in this field are also being formulated.

The Committee is exceedingly hopeful that in the not too distant future it can enlist the services of a technician either through some governmental agency or some major industry who will act as a fact-finder for the Committee. The volume of work on the possible developments of important phases in the field of Industrial Ophthalmology forces on the Committee the realization that many times over the amount of information *could* be gathered and made

available to the Academy membership if such technical lay assistance on a full time basis were available. The utmost cooperation has been manifest by many groups to whom the Industrial Committee has directed inquiries and problems, such as the National Society for the Prevention of Blindness, the United States Public Health Service, the Industrial Health Committees of various state medical societies, individual industries, etc.

HEDWIG S. KUHN, M.D., *Secretary*
Joint Committee of Industrial Ophthalmology

Conservation of Hearing

Preliminary studies reveal there are many problems for investigation by the Committee on the Conservation of Hearing, some of which are:

1. To promote active interest in the conservation of hearing.
   - A. Special studies of specific otological diseases.
     1. Otosclerosis.
     2. Syphilis.
     3. Congenital deafness.
     4. Tuberculosis.
     5. Acoustic neuritis.
     6. Hyperplastic otitis media.
     7. Acute and chronic suppuration of the middle ear tract.
   - B. Testing of school children.
     (Formulation of state programs to promote its adoption by encouraging sound state legislation.)
     1. Prevention.
     2. Early discovery.
     3. Treatment.
     4. Educational adjustment.
   - C. Testing of adults.
     1. Industrial fields.
        a. Prevention.
        b. Early discovery.
        c. Treatment.
        d. Educational adjustment.
     2. Routine health examination.
        a. Prevention.
        b. Early discovery.
        c. Treatment.
        d. Educational adjustment — occupational adjustment.
     3. Testing of individuals who have served in the Armed Forces with special reference to treatment, rehabilitation, etc.
   - D. To endorse and support the work of the Council on Physical Therapy, especially in regard to:
     1. Standardization of instruments for testing hearing acuity, hearing aids and other acoustical apparatus.
     2. Ethical standards of manufacturers and salesmen in the sale of hearing devices to the public.
   - E. Education of physicians, public, educators and legislators regarding the problems of the hard of hearing.
   - F. To encourage individual research in regard to the problems of the hard of hearing.
   - G. Investigation and reorganization of undergraduate and postgraduate education in otology.

Presented at the Forty-Seventh Annual Convention of the American Academy of Ophthalmology and Otolaryngology at the Palmer House, Chicago, Illinois, October 11, 1942, Dr. Dean M. Lierle presiding.

INTRODUCTORY REMARKS

D. M. LIERLE, M.D.
Iowa City, Iowa

The Committee on the Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology is sponsoring a program for the diagnosis and treatment of hearing defects in school children. It is their desire to formulate a practical plan which can be adopted by the various states or local committees.

The need for such a program is evident when, according to various estimates, of the 50,000,000 school children in the United States, 60,000 at least are totally deaf, 3,000,000 are hard of hearing and 4,000,000 have speech defects which in a large part are due to impaired hearing. It is said that from one to three children have defective hearing in each school room. Five per cent or 2,500,000 school children in the United States have some degree of impaired hearing.

Some work has been done in the detection of defective hearing in school chil-

dren. Except in a few instances this has been on a small scale and little has been done in the detection or treatment of earlier types of hearing impairment.

Incomplete surveys have been made in Iowa, but little has been done as far as remedial measures are concerned with the exception of the excellent work that is being done by the Iowa State School for the Deaf.

The desire of the Committee, as stated above, is to suggest a plan which may be adopted by the various states with the following purposes:

1. Routine testing of all school children at once and thereafter in the third, sixth and ninth grades.

2. To bring all cases with defective hearing to the attention of the physician for treatment when indicated. Sufficient emphasis has not been placed on remedial measures to arrest the progress of deafness.

3. To provide special education for those with hearing losses which are not sufficiently severe to necessitate institutional training.

4. To detect all cases with severe hearing losses that require training in the school for the deaf which provides vocational training, rehabilitation and eventually employment for children as they grow into adulthood.

Certain publicity should take place before submitting a bill to the legislatures of the various states. The problem should be presented to the various county and state medical societies, service clubs, American Legion, parent-teachers' associations, school officials and the public in general.

Following this a bill should be prepared by the legislative committee of the state medical society with the cooperation of the Department of Public Instructions and the Boards of Education. The bill should contain specific information in regard to the funds necessary for salaries, equipment and travel expense. Economy should be advocated. The bill should, in our opinion, appropriate funds for testing of school children in public schools, and most important provide for referral to private physicians and clinics those cases which may benefit from treatment. Cases with more marked hearing losses should be referred to an advisory committee for disposition in regard to education and rehabilitation.

For a state with a population of 300,000 school children, the following organization is suggested:

The personnel to consist of a director, five technicians, secretary and state agent. An advisory committee would consist of the director, state agent, superintendent of the State School for the Deaf, at least two otologists, instructor in speech, director of Public Health and two lay members. The duties of this committee would be to review and make suggestions for those cases referred for educational rehabilitation.

The testing should include all children in the third, sixth and ninth grades and other special cases brought to the attention of the director. The tests should be given, as a minimum in these grades, at least every three years in each local school unit and more often if possible.

The method of operation might be as follows: A schedule formulated by the director. The superintendent of the local school unit would be advised at least a month in advance of the time of the tests. The local county medical society would appoint an otologist to conduct the physical examination. The school officials and school nurse would assist in the examination. At the appointed time the audiometric technician would arrive and select a suitable place for the examination. Following the examination the cases with definite hearing impairment could be referred to the examining otologist. The parents would accompany the child in order to give an adequate history and for an explanation of the examination. Those cases which are considered remedial would be referred to the general practitioner who in turn would refer the patient to an otorhinolaryngologist. Indigent cases would be referred to the various clinics. Cases with more marked hearing losses would be referred to the advisory committee who would recommend private or public schools for special instructions.

It would be the duty of the state agent or social worker to keep a careful follow-up of all cases with impaired hearing.

No attempt has been made to furnish specific information in regard to the necessary funds, technique or testing, et cetera, as this is being compiled at the present time and will be presented later.

Symposium: The Detection and Treatment of Impaired Hearing in School Children

THE ADMINISTRATION OF AN ORGANIZED PROGRAM

HORACE NEWHART, M.D.
Minneapolis, Minnesota

Planned school health education and the early application of corrective medical care during school life offers large possibilities in the attainment of higher national health standards. The inclusion in the school health program of a definite plan for periodically testing the hearing acuity of all pupils by modern methods has made the school the most important unit for the conservation of hearing on a nation-wide scale. The procedure has been in successful operation as a feature of the state school health program in several commonwealths including New York, Pennsylvania, Oregon, Indiana and Washington, and independently in an increasing number of communities without the stimulus of legal compulsion. The high incidence of significant hearing impairments among school children and the advantage resulting from their early discovery when followed by corrective care has been fully demonstrated by the mass-testing of several million pupils by the audiometer.

It should be emphasized that the objective of the tests is not only to disclose those pupils who already have recognized or suspected hearing handicaps. Of even greater importance is the discovery of the larger number of children who have slight, often subjectively unnoticed but potentially significant hearing deficiencies, that they also may be given prompt corrective medical care and educational adjustment.

Unfortunately the need of greater familiarity with the fundamental principles involved in carrying out this program, a lack of uniformity of procedure, standardized technics and criteria, accurately calibrated equipment and a shortage of trained testing personnel have delayed the wider adoption of modern periodic hearing tests by school officials.

The urgent need of a practical, simple, economical state program for conserving the hearing of school children to serve as a model for school and public health executives is obvious. Effectively administered such a program will give to all pupils who have existing or threatened hearing deficiencies the benefit of discovery at the time in life when corrective care will yield the best results.

The American Academy of Ophthalmology and Otolaryngology is giving serious consideration to the formulation and promotion of a practical program which will include the minimal essential provisions which may be adopted in our several states, and, with modifications, in urban and rural school units where a state program is not in operation.

A state plan for the early detection and care of pupils who have hearing deficiencies will be most successful when its administration is legally made the joint responsibility of the state board of education and the state board of health working in the closest cooperation. These agencies, since the problem is largely medical, must have the understanding cooperation of the medical profession as represented by the state medical association.

The functions of each group are closely related but distinct. The board of health will establish the necessary standardized technics for the discovery and interpretation of hearing deficiencies and will prescribe the procedures for securing the best corrective medical followup of pupils found to have hearing impairments. The last function must be accomplished without invading the private practice of medicine, nevertheless insuring that all children with defective hearing will receive the care of licensed physicians especially qualified to treat ear diseases. Provision must be made that children of parents in the lower income brackets be given equally skillful care through local or state clinical and welfare agencies.

The board of education with the board of health will be responsible for the application of the tests, the notification of parents of the findings and the recommended procedures for the care of pupils discovered to have hearing impairments and the proper recording of the results of the medical and educational follow-up.

To the board of education belongs the specific duty of providing the indicated educational adjustment for all hearing deficient pupils; also definite instruction of pupils, teachers and parents concern-

ing the preservation of the hearing. The board of education will stimulate interest among state and local medical and parent-teacher groups in the prevention of avoidable hearing loss.

A further function of the state boards of education and health is to provide adequate instruction of public health nurses, school nurses and technicians in the proper technics of making audiometer tests. This is best accomplished by short, practical courses in state universities and teachers' colleges with certification of proficiency issud on satisfactory completion of a course.

The detailed administration of the state school health program for the conservation of hearing should be delegated to a qualified full-time or part-time executive officer or Technical Supervisor of Hearing, preferably a person with a medical and educational background. He or she will be appointed jointly by the state board of education and the state board of health subject to the approval of the president of the state medical association. He will direct the necessary technical staff and be responsible for the care, upkeep and calibration of the state-owned apparatus used in conducting the tests. His office will provide a uniform system for recording and filing the results of the tests and of the follow-up of all pupils found to have significant hearing impairments.

The following recommendations are made by the Committee on the Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology:

1. It shall be required by legislative enactment that every pupil in a public, parochial or private school within the state be given scientifically approved hearing tests as prescribed by the state board of health and the state board of education at the time of enrollment, or as soon as possible thereafter and subsequently at such intervals as may be designated by the above named agencies. A hearing test and an otological examination, if indicated, shall be made of any pupil on the request of the teacher, parent or guardian; also following absence from school on account of illness which might involve the hearing. A pupil may be excused from such test and an otological examination only on the written request of a parent or guardian.

2. Adequate funds must be specifically appropriated for the use of the state board of health and the state board of education for initiating and carrying on the school program for conserving the hearing.

3. The above agencies must be given legal authority to enforce the law and such rules and regulations as they may make for the effective administration of the program.

To insure accuracy of performance the audiometers used should be those "accepted" by the Council on Physical Therapy of the American Medical Association.

It is recognized that screening tests for case-finding purposes are more accurate when made by the pure tone audiometer using the sweep-check method than when made with the more rapidly applied group phonograph audiometer. In communities with large school populations the screening tests by the latter type of instruments if already in use will probably be continued as a matter of economic necessity. When this is done it should be with full recognition of their known limitations: 1. They do not disclose hearing losses for the higher frequencies now recognized as very important. 2. They are not suitable for economically and accurately testing the hearing of pupils too young to write numbers dictated by the fading voice. These are of the age when early discovery and timely care are most effective.

It is the recommnded practice before referral for an otological examination to check with the pure tone audiometer all pupils found to have hearing deficiencies by screening tests made by either type of audiometer. Therefore, in securing new equipment the pure tone audiometer is preferable for school work because of its applicability in testing all age groups.

Outright ownership of acceptable audiometers has many obvious advantages and should be required in all local school units in which there are 800 or more pupils. In small schools with limited budgets and undeveloped health programs equipment and operating personnel must be supplied by state agencies.

The cost of initiating and administering an economical, effective state conservation of hearing program is a matter of early concern to the taxpayer. No data are available for a close estimate of the per capita cost in different states. The

cost depends upon many variable conditions as the size of the school population, its distribution between urban and rural areas, the degree of development of the existing school health program with reference to available personnel for carrying out the hearing tests and follow-up with a minimal additional expense, the item of transportation in rural areas, the intervals between the periodic tests of all pupils and the charges assessable directly to the local budgets and those chargable to state funds.

In general, the cost of initiating and administering a school conservation of hearing plan is less in states and cities which already have an organized health program. Larger cities are usually well able to meet the additional expense of including adequate hearing tests in their programs. Smaller school units in rural areas require generous help from state tax-raised funds. It is therefore recognized that larger communities may justly be required to meet the cost of the hearing program. To insure uniformity and effectiveness in its application all tests should be under the direction of the State Supervisor of Hearing.

In certain states the law requires annual hearing tests of all pupils. It is the opinion of many otologists and educators that tests at such intervals are unnecessarily frequent and impose too great a burden on the taxpayer. It is recommended that the periodic tests after the one made at the time of enrollment be routinely made of all pupils during their fourth, seventh and tenth school years. Selected individuals are given special tests as may be indicated by their scholastic achievement, behavior and health records.

Recent figures from the State of Indiana indicate that the annual cost of the screening and confirmatory checking tests are covered by twelve cents per pupil on a basis of 30,000 individuals in a given geographical district.

To initiate and maintain an adequate state program there is needed an estimated annual budget of from $10,000 to $30,000 roughly allocated as follows:

Administrative Costs

Salary of the Supervisor of Hearing	$2,500 to $4,500
Secretarial help supplied by the state boards of health and education	500 to 1,000
State owned audiometers, cost to be amortized over a five-year period, annual charge	1,000 to 2,000
	$4,000 to $7,500

District Operating Unit Costs (for One Unit)

Trained nurse or technician for each district having a school population of 30,000 individuals, salary	$1,500 to $1,800
Local helpers on hourly basis	300 to 400
Traveling expenses	250 to 500
Mileage at legal rate	200 to 250
Supplies	50 to 60
	$2,300 to $3,010

The total annual cost during the first five years will depend on the size of the school population, allowing one operating unit for each 90,000 pupils since the periodic tests are made on each group every third year. In compact city schools the cost will be materially less than in rural areas.

The cost of incorporating such a program is more than compensated by the saving to the state in taxes for the special education of needlessly handicapped pupils and the support of individuals made unemployable by severe, avoidable hearing losses.

TESTING HEARING OF SCHOOL CHILDREN

HOWARD A. CARTER, Ph.D.
Secretary, Council on Physical Therapy, A.M.A. (by invitation)

Plan of Execution

Need for Plan

Although the testing of hearing in school children has been carried out in many cities and in several states, organized and singleness of purpose on the part of the medical profession is strictly

new in this field. The time has now come when otologists, as the leaders, with the cooperation of all other branches of the medical profession, should direct the activities in testing hearing of school children. A universal plan which would be acceptable to all of the present groups now functioning in all states, counties and cities should be formulated and adopted. It is highly probable that many of those that are testing hearing now in certain localities will find it necessary to revise or improve the method if they have any notion of cooperating.

Publicity

Publicity is neded. Superintendents of schools, state, county and city, principals and especially the teachers of the lower grades should be approached with a comprehensive program for attacking this problem. Parent-teacher organizations, national education societies and local educational groups, both sectarian and public, should be reached.

Modus Operandi

How to reach the important people to obtain this end is a matter that needs careful consideration. Better instruction within the ranks of the medical profession is probably the most important starting point. Instructors in otology should dwell on the problem of testing hearing in school children. Deans of the medical schools should be approached with the idea in view of reserving a portion of the curriculum for this important purpose. To inform the pre-medical student, doubtless the most effective way to influence the medical curriculum is through the Council on Medical Education and Hospitals of the American Medical Association and through the Association of Deans of Medical Colleges. Practicing physicians and leaders in the medical profession may also be reached by papers given before state, county and city medical societies and before the special societies such as the American Academy of Ophthalmology and Otolaryngology, the American Laryngological, Rhinological and Otological Society, and the American Otological Society.

Influence from without the medical profession may be obtained by presenting papers on the subject to the National Parent-Teachers' Association, as well as the state and local associations, and also the national, state and local educational groups. In view of the activities of the American Society for the Hard of Hearing, which has a pretty good program already prepared, there will be no difficulty in obtaining cooperation from this society.

Influencing Important Individuals

How to interest and influence key individuals after they have been reached is a matter that needs most careful study. Although the reading of papers on the subject has been tried and in many instances has been successful, a more energetic program is recommended. Exhibits utilizing posters, etc., have also been used with considerable success, but here again improvement is needed. Premedical instruction in courses on otology dealing with the importance of prevention of hearing loss is vital in beginning a long range program and as soon as a comprehensive method is compiled this important source of influencing modern viewpoints should be tapped. Postgraduate or refresher courses in clinics will have a powerful influence on the physicians who are looking for ways and means to better their position in the community.

Exhibit

Your Committee visualizes a large exhibit to be given at large conventions, such as the annual session of the American Medical Association, the American College of Surgeons, the American College of Physicians, the Catholic Hospital Association, and possibly the National Association of Education and the Parent-Teachers' Association. This exhibit will be along the lines of those already carried on by the Committee on Fractures and the Committee on Low Back Pain. Such exhibits are subsidized and must be well thought out before any request is made for funds from any organization such as the Board of Trustees of the American Medical Association.

A quiet place in the scientific exhibit can be chosen, a stormsash borrowed from local lumber yards and the room made as quiet as possible. This enclosure should be approximately 40 by 50 feet, carpeted, and drapes hung to minimize sound intensity and prevent reflection of sound. The audience would congregate outside, sitting on benches such as used in a circus. Instruction and explanation

on the demonstration being carried on inside of the quiet room shall be announced through loud speakers outside of the enclosure while the instructor moves about with a microphone on the inside. Several demonstrations can be staged in one day. Cooperation may be secured from the local school authorities, and several groups of children tested by means of the approved testing instruments such as phonograph-group audiometers and the pure tone group audiometer. Those students found to have a hearing loss may be examined in another portion of the enclosure according to the minimum examination plan which will be decided upon later. Different instructors will be chosen for each forenoon and afternoon and still different ones for the number of days of the meeting. A new group of children should be used at each demonstration. The entire exhibit should be under the control of a committee appointed for the purpose.

The advantages of this method for showing the importance of testing school children are as follows: It attracts the attention of the general practitioner and physician leaders in other fields. The "living" aspect of this kind of exhibit has a greater appeal (showmanship) than does a static exhibit with charts. The importance of testing a child early is impressed upon the general practitioner. The most important reason for such an exhibit is the fact that specialists in otology are called in from different parts of the country, given an opportunity to demonstrate, get their names on programs and become interested in the field because they are part of the program. Another valuable point, but not usually mentioned, is that the instructors reach common viewpoints and a specific program emerges while individual and personalized opinions are eliminated because of the weight of opinion which emerges in the broad acceptable forward movement.

Such a comprehensive exhibit cannot be put on without considerable ground work and study. A committee should experiment first. One place to start would be in the scientific section of the American Academy of Ophthalmology and Otolaryngology. The "bugs" in an exhibit of this kind can be ironed out and it can be made to function to the best interests of all concerned by a less expensive means, e g., utilizing quiet rooms in hotels.

Adjoining or nearby the aforementioned exhibit might well be an exhibit of the American Society for the Hard of Hearing, in which demonstrations and contests are given in lip reading. In large cities such as New York and Chicago there are many lip readers and in fact means might be provided for offering prizes for the best lip reader of the day or the week. Physicians themselves might volunteer to do the speaking, thereby bringing in more interest on the part of the on-lookers. At the present time there is insufficient publicity among the general practitioners to inform properly their patients regarding the value of studying lip reading not only for the deafened child so that he can take his proper place in society and in school, but for the adult who is gradually growing more and more deafened.

AN EDUCATIONAL PROGRAM FOR THE DETECTION, PREVENTION AND AMELIORATION OF IMPAIRED HEARING

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Deafness and defective hearing constitute one of the major problems of preventive medicine and public health and present a definite challenge to the medical profession. In the solution of this problem the profession has been somewhat laggard and dilatory. Inability in the past to improve greatly the fixed hearing defects of adults and lack of interest in detecting early defects in children have produced a policy of "laissez-faire." Physicians must be awakened from their lethargy and imbued with the thought that much can be done — particularly in the early detection and prevention of the trouble. Just as special cults have removed large numbers of the people from good medical attention because the physician refused to regard their ailments as a serious medical problem, so audiometrists and salesmen for hearing aids are gradually but surely taking the play away from the otologist because he refuses to concern himself with something for which medicine and surgery, as formerly practiced, offers no

ready solution. True, here and there isolated physicians have been alive to their responsibility and have struggled to make a dent in this enormous problem; but, by and large, it must be said that physicians as a whole have been indifferent. This gigantic problem must be vivified, unified and nationalized under the control and direction of the medical profession.

The American Academy of Ophthalmology and Otolaryngology, through its Committee on the Conservation of Hearing, for the detection, prevention and amelioration of impaired hearing, has a very vital interest in this program and is attempting by education, organization and liaison with other public bodies to stimulate and vivify the interest and cooperation of the medical profession and the public at large in this hitherto somewhat neglected aspect of preventive medicine.

Realizing that defective hearing in the adult is largely an irremediable condition in that it has become fixed and usually not amenable to medical or surgical treatment, and realizing also that the reverse is much more often true in the child, it behooves us to concentrate our efforts for the detection, prevention and amelioration of this condition in the children of elementary and secondary school age. Recent surveys made in various parts of the country indicate that hearing defects, serious enough to demand investigation and treatment, are present in from 2 to 10 per cent of the entire child population.

The detection of these hearing defects in children is not an insurmountable task. The main bottleneck in the past has not been the detection of the hearing impairment, but rather its prevention and treatment. A detailed plan for the follow-up work in prevention and treatment will be taken up by another member of this Committee.

Congenital defects, cerebrospinal meningitis, scarlet fever, mumps and measles account for about 10 per cent of the hearing defects of childhood. These conditions produce a type of hearing defect which is often not amenable to treatment. However, even these children can be protected against a further deterioration of their hearing by a campaign against such conditions as otitis media, lymphoid hypertrophy in the nasopharynx and frequent colds, which tend to superimpose a conductive type of deafness upon their pre-existing nerve lesion.

The remaining 90 per cent of the impaired hearing in childhood is largely preventable and remediable by an active campaign to prevent the spread of colds among children, to limit the overgrowth of lymphoid tissue in the nasopharynx — especially in Rosenmuller's fossa and about the tube mouth — to lower the incidence of otitis media, if possible, to encourage its early detection and vigorous treatment and to improve the general health of the child. The most poorly performed operation today is the adenoid operation. Pediatricians, general physicians and even otologists must be taught that, for the prevention and cure of deafness in children and adults, it is much more important to do a clean, thorough adenoid operation than to remove the hypertrophied tonsils. It is not enough merely to remove a mass of adenoid tissue and call the adenoid operation complete. Under direct vision and certainly by the use of the exploring finger it must be determined that every macroscopic vestige of lymphoid tissue in Rosenmuller's fossa and about the tube mouth has been removed. This, together with the early detection and vigorous treatment of sinus infection, will help greatly to reduce the incidence of otitis media. To attain this objective the profession must have the earnest cooperation of the health agencies and school authorities of city, county, state and nation.

Systematic examinations for the detection of beginning hearing impairment should be made in the schools at least by the time the child has reached the third grade and should be repeated at stated intervals. The defect can be picked up at an even earlier age by parents, nurses and teachers if their attention is focused on the problem. If the parent, nurse or teacher suspects a possible hearing defect in a child below the third grade a pure tone audiometer screening test can be made in even these younger children if they possess average intelligence. Such a program requires the education, active interest and cooperation of parents, school boards, school physicians, nurses and teachers under the guidance and direction of the otological profession.

In the solution of this vital program the interest and cooperation of the entire

medical profession is essential. This can be encouraged and stimulated by an educational program for the medical student, the otologist and the general medical practitioner. Unless the interest and cooperation of the medical profession as a whole can be elicited, the program will fail.

Because of the crowded curriculum in our medical school courses the specialities are restricted to minimal requirements, and yet I am convinced that some instruction of medical students on the detection and prevention of hearing defects could be included in the course on otolaryngology if the deans of medical schools and the heads of departments of otolaryngology were systematically contacted and impressed with the importance of the problem. As the head of such a department I am convinced that such instruction would be much more valuable to the medical student than some of the more intricate phases of otology which are being taught today. Many of the teachers of otology for undergraduate students lose sight of the fact that they are teaching otology to the undergraduate and not to the embryo otologist. Only those phases of otology which will make a better practitioner, both urban and rural, are necessary for the undergraduate student.

As to the specialist himself, I think I can say without fear of contradiction that there are no better trained and more competent otologists in the world than exist in this country. And yet, I think it must be admitted that with a few notable exceptions the otologist of this country is not particularly interested in the problem of the detection, prevention and amelioration of defective hearing. He is not particularly interested because, in spite of the recent advances in the field of prevention and in the field of treatment or amelioration of hearing defects, he is still largely imbued with the old idea of the futility of trying to do anything for this condition. He must be impressed with the fact that in any teamwork to handle this subject he is the main cog; that without his interest, help and cooperation the whole program must fall by the wayside; that the nurses, teachers, technicians and other personnel must be taught, guided and supervised by him, and that in the treatment of the conditions mostly responsible for hearing defects in children he is the court of last appeal. If he sidesteps this issue, he is untrue to his trust.

To the end that the otological profession should become aroused to the importance of this issue, an effort should be made to interest especially the secretaries of all otological or otolaryngological societies to put on their programs from time to time papers dealing with this subject. Only by hammering away at this subject will the lethargy of the otologist be broken up. Only in this way will he be aroused to a sense of responsibility in this field.

For the education of the profession at large and also of the general public, I would advise the formation, under the egis of each state medical society, of a Central State Committee on the Conservation of Hearing, such a committee to consist of prominent physicians, prominent otologists and prominent laymen—particularly those interested in public health and education. It might be advisable for the Academy to appoint an otologist in each state to initiate such a state medical society program and to help set up the necessary machinery. Such a general committee could then organize similar subcommittees for each county or each councilor district, as the case may be.

If the public can be impressed with the fact that, in addition to the health program involved, there is an economic aspect and that large sums of money now needed to provide special education for the hearing defectives can be saved, I am sure that the enthusiastic support of the public can be expected. If such state committees functioning under the general direction of the Academy Committee could be formed, I am sure that headway could be made.

Such committees could through the press, radio and public gatherings make the people realize that the project has both educational and economic aspects in addition to the public health problem involved. School boards could be made to see that there would be a great saving of public monies if the incidence of defective hearing could be cut down. Figures must be available as to how much it costs to educate the hearing handicapped child in special schools for the deaf and the hard of hearing.

There must also be a large economic loss from defective hearing in the adult population for there are many industries in which these people cannot work advantageously. By cutting down the incidence of childhood hearing defects we are also going to cut down the incidence of adult non-effectives. We will save large sums of money in the education of the young, and we will conserve the economic value of the adult population. Surely these are worthy objects in addition to improving the health of the population.

The medical profession can interest the public as personified by the school authorities and parent - teacher associations in the wider use of hearing aids by children. This applies to those children whose handicap is so marked as to necessitate education in special institutions at high cost to the taxpayer. These handicapped youngsters could then be educated in public schools at far less cost, and this would prevent their seclusion and segregation with the resulting development of an inferiority complex such as our present system is bound to develop.

The use of hearing aids by handicapped school children is a subject which has not been developed up to the present time, but is one to which we should give serious consideration. A youngster with a hearing aid can be educated along with his fellows and grow up with a normal mental viewpoint, which is not true if he is secluded from his playmates and segregated in a special school for the deaf.

There will be those who will argue that to put a hearing aid on a school child puts a stigma on him, but I say that we are sooner putting a stigma on him when we segregate him in special schools for the deaf. For that matter, the school child used to be stigmatized when he had to wear glasses, but gradually usage has obviated all that.

If school boards cannot be interested in putting through a program for the detection, prevention and amelioration of deafness, or in helping to provide the needy child with a proper hearing aid, I am sure that such agencies as Junior Leagues, the American Legion, the various service clubs and the like can be interested in financing such projects.

I am, however, convinced that we have approached this problem from the wrong angle in the past. School surveys to seek out the children with deficient hearing are very meritorious and have furnished us with valuable information on the incidence of these handicapped children in our schools. The first procedure in any constructive program must deal with the matter of detection; but, unless adequate provision for the care and treatment of these juvenile cases has been set up, it is useless to attempt any further surveys. Following the enthusiasm which originally greeted these surveys, the profession and the public have developed an apathy for the whole subject because no adequate and systematized machinery has been set up for the treatment of these children. The method of making these surveys has been pretty well developed. The next important problem before physicians, educators and the public is to develop ways and means for the treatment, guidance and care of the hard of hearing child. Until this problem has been worked out, it is useless, in my opinion, to spend any more time, effort and money in the matter of detection.

PUBLICITY

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The purpose of this brief thesis is to discuss how this great Academy with its valuable facilities for education can aid the movement sweeping the country with such cumulative force in the discovery and aid to the child with defective hearing. This must be done with ample credit to the indefatigable efforts of the pioneers who have already put this upon its feet. The importance of correlating methods of approach, obtaining uniform standards to technic and adequate working equipment is obvious, and it is timely for this body to enlist the service effort of the efficient group of otolaryngologists in its membership.

Your Chairman has emphasized the importance of intelligent publicity to obtain a uniform, country-wide organization. This organization should recognize all important phases of defective hearing and lend its aid toward practical research in the conservation of adult hearing. This concerns:

1. In the pre-school age:
   a. Methods of prevention of hearing defects.

b. Recognition of early symptoms of hearing defect.

c. Management of hereditary and partially hereditary defective hearing.

2. In the school age:

a. Early and accurate screening of children with defective hearing.

b. Cooperation of parents, teachers, private physicians with the school health and education departments in keeping doubtful cases under observation.

c. Provision for definite arrangements in placing those with serious handicap under otologic attention.

d. Adequate procedures for obtaining instruction in lip reading and proper hearing aids for irremediable cases and rehabilitation in the economic usefulness of today.

3. In the adult age:
Medical and surgical therapeusis. Biochemic reconstruction. Education in the hygiene and conservation of hearing during the aging process.

It is high time for the distinguished group of otologists forming the Academy's Committee on the Conservation of Hearing to cease its annual conference function and become an activating force in harmonizing and standardizing the splendid work which has already been done in various sections of the country. An enormous amount of literature on hearing deficiency has been published in the last decade, mostly dealing in vague generalities and not conducive to any practical uniformity of procedure. Such is absolutely important if national and state legislatures are to be tuned to its importance and direct their departments of health and education to carry out effective discovery and remedial measures for deafness. Its social, economic and vocational importance should need no brief.

These matters should principally concern and perhaps in this order:

1st—Parents, relatives and immediate friends of those with defective hearing.

2nd—State departments of health and education, with the complete cooperation of national, state and county medical societies.

3rd—All national and sectional societies and organizations whose chief concern is otology.

4th—Business, industrial and professional organizations, both for their dependency upon good hearing and for guarding occupational deterioration.

5th—Private and institutional agencies engaged in the economic and vocational rehabilitation of those with complete or partial hearing deficiency through lip reading and hearing aids.

These groups must all be stimulated into cooperative help in carrying out standardized procedures, whose requirements are obviously:

1st—An accurate program.

2nd—Adequate and standardized equipment.

3rd—Skilled administration.

Final Comments:

So much for the pattern in the background of this paper. Now let us look the facts in the face and see what should be considered as comments or conclusions:

1st. Inasmuch as legislators who authorize and direct the departments of education and public health are always keenly responsible, from political reasons, to the majority wish of their constituents, steps must be taken to get the parents of these children with hearing deficiency sufficiently ear conscious to demand these appropriations in the various states.

2nd. As somewhat less than 10 per cent of children of school age are deficient in hearing, efforts must be made to dramatize and advertise the possibilities of hearing deficiency and its consequence, in order that the spirit of protective anxiety may be vividly aroused.

3rd. Legislative and parental influence would lay the ground work for stimulating these otologic associations and foundations with allocatable funds. This would be done through skillful individual and committee contact and perhaps special Federal blessing.

4th. National, state and medical organizations should be made keen to cooperate in this service, provide the refresher

education to the somewhat indifferent average physician in teaching him the diagnosis of otitis media, portraying the etiological factors and therapeutic measures, and finally provide the expert otologic avenues for diagnosis and treatment of demonstrated and difficult ear disorders.

5th. You are well aware how infantile paralysis, tuberculosis, venereal disease and blindness have fastened the imagination of the public through skillful dramatization and advertising. Why shouldn't this be applied to the child struggling to hear, through press, radio, pulpit, forum, women's federations and clubs, parent-teacher associations, service clubs and motion pictures?

6th. The wartime restrictions should be overcome when it is pointed out how the aggressor nations have set the pattern in rearing many and perfect children.

Above all, let us recognize that the Academy is simply following on and centralizing much of the great work in this field which has been accomplished in so many states. We should not antagonize, arouse jealousy or otherwise hinder, but rather stimulate closer association with our body of great minds who have already done such grand work in this field.

THE MEDICAL ASPECT OF THE HARD OF HEARING WITH ESPECIAL REFERENCE TO TREATMENT

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This subject should remind us that our ears are not a separate entity from the rest of our body, but one of the integral parts of our whole mechanism, and they are, therefore, subject to any and all changes to which the human body is heir.

It should be taken into consideration what methods are used to reveal the condition; that is, the examination of the child, the interpretation of the findings, the treatment to be employed in relieving the condition and the advice as to the future care of the patient.

The method of discovering these hearing losses has been discussed by other members of this symposium. Once this hearing loss has been established we ask, "What disposition of the case should be made?" Those in charge should contact the parent or parents of the child and should explain to them the importance of further investigation of this defect. They should ask the parents to get in contact with their family physician and to discuss the problem with him. In most cases the family physician will, in turn, refer the case to a proper otologist who will make an examination of the ears, endeavoring to discover the type of hearing difficulty from which the child is suffering and reporting the findings to the proper authorities on forms which are furnished to him for the purpose.

A time limit should be set for the return of this form, and if it is not returned in from four to six weeks the family should be contacted to find out whether the child has been examined, and if not, why not. (Of course, some school systems have their own examining otologists and the examination is made and filed at once, the child is sent to his family physician and on to a practicing otologist of his choice for treatment.)

When the problem of the hard of hearing is discussed relative to the medical aspect it naturally must include the following: (1) prevention, (2) examination, (3) diagnosis, (4) treatment and (5) rehabilitation.

It is generally agreed that if we are to prevent hearing difficulties it will be necessary to discover the causative factors producing this condition at an early period in the child's life. If it were possible to have every child examined before the age of three years, we might be able to discover the hearing defect and to remedy the etiological factor early, thereby, at least limiting the condition, and, in many cases, returning the hearing to normal. Perhaps the most usual indications for examination and treatment are earache, lack of nasal respiration and inattention of the child to its surroundings.

A discussion of the pre-school age child is not a part of this symposium other than to bring out the point that an early discovery and diagnosis of a hearing defect will lessen the number of cases which will be discovered after school age.

When a child reaches school age he is supposed to have normal hearing. Experience has proven otherwise, however, and it is by a complete check-up of the

hearing of all school children, by methods already described by other members, that those with defective hearing are discovered.

When the child with defective hearing is referred to the otologist for examination the first and an important factor in arriving at a diagnosis is for him to take a complete and careful history of the case. A carefully taken history brings out many bits of information which are usually overlooked in a loosely taken history.

After having taken the history, and in order not to bias our opinion in any way, it is better to make the functional hearing tests before a clinical examination is made.

These tests should include the following: An audiometer reading, with a recorded graph of each ear; tuning fork tests, i.e., Webber and Rinne, and conversational tests with both spoken and whispered voice. These should be conducted in a room which is, as nearly as possible, sound proofed.

After these tests are completed and noted, we should continue with the clinical examinations, which should consist, first, of an inspection of the parts affected, and, second, of those regions which have to do with the pathology causing the disturbance, which are:

1. External ear.
2. External auditory meatus.
3. Membrana tympani.
4. Nasal cavity.
5. Postnasal space, including choana.
   a. Adenoid, mouth of internal auditory tube, fossa of Rosenmuller.
6. Pharynx.
   a. Postpharyngeal wall.
   b. Lateral lymph columns.
   c. Tonsillar area and tonsils.
7. Hypopharynx—base of tongue.
8. Larynx.
   a. Glottis and functioning of vocal cords.
9. Laboratory findings, especially a urinalysis, blood picture and X-rays when necessary.

If, on examination, the external auditory meatus reveals blocking by atresia, exostoses, impacted cerumen, inspissated pus, or by an acute, subacute or chronic inflammatory condition, they should be remedied. If, by the inspection it is revealed that the external meatus is normal, the membrana tympani should be carefully inspected and any deviation from normal noted. Some points to be noted are the color and landmarks, whether there is retraction, thickening or other findings which indicate repeated inflammatory attacks. It should be noted whether there are any perforations in the membrana tympani, and if so their location. It also should be observed whether or not there is any discharge coming from the middle ear. The character and odor of this discharge should be studied. These are significant of the existing pathology.

An examination of the nasal cavity should reveal any intranasal blocking due to deviation of the septum, and it should reveal any neoplasms or evidence of sinusitis. If any of these exist it is necessary to correct them in order that the patient may have proper aeration of the middle ear and to remove the causes for recurrent infection of these parts.

Hearing losses are usually classified as of three major types, (1) the conductive type, (2) the perceptive type and (3) the mixed type (which is a combination of the first two).

In making our diagnosis the above classification should be followed.

The conductive type of defective hearing is far more prevalent in school children than any other type, and it, of necessity, should hold more interest in this discussion. It is important that a study of the pathology and areas affected should receive considerable study.

If the hearing loss is found to be of the conductive type the treatment indicated will be governed by the type of the pathology found. The most common etiological factors in producing a conductive type of deafness are to be found in the postnasal space, either in the eustachian tube itself or about the mouth of the tube in the fossa of Rosenmuller, or in the adenoidal area.

Recurring inflammatory changes due to infection in this area produce a hyperplasia of the mucosa and an increase in the size and amount of lymphoid tissue with adhesions between the posterior lip of the tubal orifice extending across the fossa of Rosenmuller to the posterior wall of the postnasal space.

In a general way treatment should be directed, as nearly as possible, toward disposing of the etiological factors concerned in the condition.

As it has been stated, the conductive type of hearing difficulty is the one most commonly found, and in this condition the epipharyngeal area is the one in which the causative factor is most commonly located. Our first effort should be directed toward correcting the conditions found in this region.

The most common pathological findings in this area are excessive lymph tissue and hyperplasia of the mucous membrane.

There are two important methods of remedying this condition, i.e. (1) surgery and (2) irradiation.

In discussing the surgery it is not necessary to go into the slightly varying technics other than to say that removal should be done in a meticulous manner and under direct inspection. If the procedure is carried out in this manner the results are usually very satisfactory.

Irradiation for the reduction of lymphoid tissue in this area has produced very excellent results in the hands of some observers, especially Crowe and Guild of Johns Hopkins.

One would be prone to think that a combination of the two would and should be of benefit at times.

There are, of course, many other conditions producing this type of hearing difficulty and they are as follows: Impacted cerumen, bony exostoses, allergy, chronically discharging middle ears, etc. The treatment will not be discussed as it is presumed that most otologists are well acquainted with the technic necessary to care for these conditions.

In the perceptive type of hearing difficulty we are confronted with an entirely different problem. A lesion may exist from the nerve ending through the entire course of the nerve to the cortical area. This oftentimes presents a problem from the neurological as well as the otological side. This type of hearing difficulty may be due to blood dyscrasia, toxemia from drugs or infections, allergy, specific infections; it may follow injuries involving the base of the skull, tumors of the auditory nerve, brain tumors and abscesses, meningitis or poliomyelitis; the acute exanthemata, especially measles, scarlet fever and mumps may be responsible for the condition.

The treatment indicated in the above types of impaired hearing is based on the findings at examination. If it is a blood dyscrasia, the blood picture should be built to normal if possible. If it is a toxemia the toxic influence should be removed if possible. If it is due to a leutic infection, antiluetic treatment should be given. Fractures at the base of the skull involving the cochlea or labyrinth will not usually improve with treatment. Tumors of the brain producing pressure in the brain stem involving the nucleus of the nerve may be removed and an improvement of the condition may occur. When meningitis is responsible for the condition usually no results are obtained.

All in all, treatment for the perceptive type of deafness is not particularly satisfactory.

The mixed type of hearing difficulty is a combination of the types just discussed and it is not so amenable to treatment, but many times we may correct the conductive portion of the trouble, which has been discussed, and have only a perceptive type of hearing difficulty left with considerable improvement in the hearing of the child.

Many times these cases require a general physical checkup and it will be found that general medical care may improve the whole picture of the hearing difficulty.

The problem of rehabilitation does not deal with those patients whose cases yield to treatment, but to those who have no chance of improvement and who indicate by the examinations that they will probably gradually go on to a complete conversational loss of hearing. It is important that a plan to rehabilitate them be set up.

If there is any phase of medical service where encouragement is needed, it is in this type of case. To say bluntly to a parent that we have nothing to offer the patient in the way of treatment is rather harsh. Of course we know that we can do nothing for these cases by treatment, but we should direct them in the course of their future activities.

They should be instructed to learn lip reading, or the manual method of communication, and should be told and instructed about hearing aids and how to

use them. Both lip reading and hearing aids may enable the children to carry on with their educational activities and make their surroundings much more comfortable and pleasant, thus preventing them from being ostracized by their friends and fellow students.

Summary

Loss of hearing should be discovered early in a child's life.

A proper diagnosis of the condition should be attempted.

The proper treatment should be instituted early if it is to be of value.

If the loss of hearing is of such a degree that it will not yield to treatment a rehabilitation program should be planned.

TECHNIC OF HEARING TESTS

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(By Invitation)

I. Introduction

The success or failure of this program in large part will depend upon the training, efficiency and personality of the State Technical Consultant in charge of the administration of the hearing tests. Since he is one of the few full-time individuals associated with all the various aspects of the program he is the logical person to serve as coordinator between the different agencies and individuals involved. He will function also as statistician and research director of the medical and educational follow-up procedures, and prepare the forms and blanks necessary to the administration of the program.

The State Technical Consultant must be well trained in the technics of hearing tests, evaluation of results, and he must know how to check on the calibration and repair of the testing equipment. Information on the fitting of hearing aids, lip-reading and the relationship between impaired hearing and defective speech is recommended. It is imperative that the major scholastic work of the Consultant consist of pertinent courses in psychology, speech, physics and physiological acoustics. Association with an otological clinic for a year if possible to obtain practical experience in the testing of hearing, evaluation of results and fitting of hearing aids is highly recommended. Such training can be obtained in various parts of the United States at the present time. This background would enable the Consultant to train and direct the number of assistants required to complete a given testing program within a desired period of time.

The Consultant initiates the program by informing the school superintendents that the testing service is available and requests the superintendent to indicate which of two or more dates he prefers, together with the estimated number of children to be tested and the number of different buildings in which the testing is to be done. This will enable the Consultant to determine in advance the approximate amount of time it will be necessary to allocate to each town. The Consultant also must contact the president of each of the county medical societies to learn the names of the otologists who are to take the history and make the ear, nose and throat examination of the children found with defective hearing in their respective localities. The otologist is then notified a few days in advance the exact date his services will be required at the specified school.

II. Equipment

A. A pure tone audiometer, accepted by the Council on Physical Therapy of the American Medical Association.

B. A Council-accepted phonograph record group audiometer with extra set of records and special calibration record.

C. Audiogram blanks, which are printed on the follow-up forms.

D. Suitable noise level meter, if the budget permits.

III. Procedure

There is a gradually increasing conviction among recognized authorities that the hearing of school children should be tested only with the pure tone audiometer. This instrument reveals high frequency losses which may be indicative of incipient pathology which are not detected by the phonograph record speech test, as pointed out by Newhart, Guild, Crowe and others. However, the ideal of pure tone individual testing may be difficult of attainment when the testing of large numbers of children is involved because of limitations of trained personnel, cost and time. The complete program must be as inexpensive and practical as possible or it is doomed to failure.

If the phonograph record group audiometer is used, this Committee wishes to emphasize the desirability of retesting every child with the pure tone audiometer who failed on two previous tests with the phonograph record group audiometer. Whenever the testing of a relatively small group is to be undertaken it is recommended that the children be tested individually with the pure tone instrument. After the program is once well established and a mass survey of the entire school population completed, it is suggested that all future retests and tests of new admissions be made only with the pure tone audiometer.

If it is not possible to test each child annually it seems advisable that the following classifications of children as a minimum be tested or retested each year.

A. All children who failed to meet the minimum hearing requirements the previous year.
B. All new children entering the school system, with emphasis on children up to the third grade.
C. All sixth grade children.
D. Children who developed discharging ears after the previous test.
E. Children who developed speech defects.
F. Children who failed to graduate to the next higher grade.
G. Any child whom the teacher or school nurse recommends be given a hearing test.

Immediately after arriving on location the Consultant or one of his assistants checks on the suitability of the room designated for the hearing tests. If the testing room is selected according to the suggestions outlined in a letter to the school superintendent, the noise level of this room should be lower than that of any other room in the building. If a satisfactory room is not available in the school system, it may be necessary to conduct the tests in the basement of a quiet church or other building away from noisy surroundings.

The following procedure is recommended for the mass testing of large numbers of children when both the phonograph record group and the pure tone audiometers are used.

A. The initial test is made with the phonograph record group audiometer.
B. Children having a loss of 9 dcb or more in one or both ears on the first test should be retested by the same group instrument on the same day if possible. Repetition of the test usually eliminates a large number of those who failed the first test.
C. All children who fail to pass the second phonograph record speech test and those with speech defects who passed the first group screening test should be given the individual pure tone audiometer test.

If the Consultant has one or more assistants, the group tests may be given by the assistants. The Consultant then follows up with the individual pure tone test at the time the otologist visits the school.

IV. Analysis of the results of the hearing tests.

For purposes of otological consideration children with hearing losses shall be divided into two groups: (1) Those requiring immediate referral and (2) Those to be deferred for observation and retesting one year later. Pending results of research now in progress, the following criteria are tentatively recommended for referral and/or deferment.

A. For referral.
   1. History indicating, or a present observable pathology, such as discharging ears, diseased tonsils, infected adenoids, sinus disease, sore throat, earache, impacted cerumen, etc., regardless of hearing acuity.
   2. Those with losses of 9 dcb or more in one or both ears as measured with the phonograph record speech audiometer and/or losses exceeding 15 dcb at 512, 1024 or 2048 cycles with the pure tone audiometer. (It has been learned by experience that a 9 dcb loss on the phonograph record speech audiometer is approximately equivalent to an average 15 dcb loss on the pure tone audiometer.)
   3. Those with a loss of 15 dcb or more at 2048 cycles who have a progressively greater loss for the higher frequencies.

B. For deferral.
   1. Borderline cases with hearing losses between 10 and 15 dcb at 512, 1024 and 2048 cycles with

the pure tone audiometer, with negative ear histories.

2. Isolated drops in the audiometric acuity curve greater than 15 dcb at 4096 cycles or higher.

V. Medical follow-up.

The parents of the children in the referral group are called in for an interview with the examining otologist before he has left the building if possible. At this time the advisability of immediate otological attention is impressed on the parents. Also a letter should be addressed to the family physician, stating the type of hearing loss or pathology present, with the recommendation that he in turn refer the child to an otologist, or in case of indigencies, to a free otologic clinic. If the parents are unable to come to the school for the interview with the otologist, the school nurse calls on them and sends the letter to the family physician. The school nurse, after learning the identity of the otologist recommended by the family physician, sends him a report of the school otologist's findings and the results of the hearing tests. Part of the report to the otologist recommended by the family physician is to be detached and returned to the school nurse after treatment. If this report from the otologist is delayed over two months, the nurse should investigate the cause of the delay and attempt to remedy the situation. If the child has not visited the otologist within the following next two months the nurse will report the case to the state agent or social worker, who will make an investigation and insist on accompanying the child to the otologist if necessary.

VI. Educational follow-up.

For purpose of educational guidance children with handicapping hearing losses may be divided into three groups.

1. Those in need of lip reading only.
2. Those in need of both lip reading and a hearing aid.
3. Those in need of individual or institutional instruction. The advisability of tutoring and speech correction must be considered in relation to every child who failed to meet the minimum hearing standards.

Although no invariable rules can be established in setting up criteria for placement in the above categories, the following suggestions serve as guides or reference points.

LIP READING. All children with an average permanent or progressive loss of 20 dcb or more at 512, 1024 and 2048 cycles.

HEARING AIDS. Children with average minimum losses of 30 to 40 dcb at 512, 1024 and 2048 cycles. The excellence of the speech, scholastic progress, behavior, I.Q., lip-reading ability, attitude in class and the ability to hear sufficiently well in class to participate in group discussions serve as practical guides or indications as to the need for hearing aids. Hearing aids are regarded as necessities for children with average losses of 40 dcb or more, provided sufficient residual hearing is present to justify their use. The minimal amount of residual hearing which would assure beneficial results from class room use of an individual hearing aid also is a variable quantity. It is doubtful if individuals with losses in excess of 85 dcb over the speech range would receive compensatory value from the use of an instrument in the average class room. In cases with extreme losses, actual trial must be relied upon for a correct estimation of the assistance that may be expected from the use of an instrument. All children in need of lip reading and hearing aids should be seated in the front of the room with the light and seating favorable for reading the teachers' and pupils' lips.

INDIVIDUAL OR INSTITUTIONAL INSTRUCTION. This group includes those with hearing losses and speech defects so severe that they cannot make near normal progress in school, even with the aid of lip reading, a hearing aid and tutoring. Private individual instruction, a day school for the deaf, or a private or state school for the deaf is recommended. All children with marked hearing losses who need private or institutional care, or who need hearing aids, shall be reported to the State Supervisor of Special Education or the State Agent. The Supervisor will give advice to the child's parents regarding various types of private and state institutions devoted to the education of those with impaired hearing, and will know how to approach charitable and service organizations for assistance in obtaining hearing aids for indigent parents of children with impaired hearing.

Responsibility for commitment to an institution for the deaf shall be vested in a committee consisting of the local school superintendent, examining otologist, State

Technical Consultant and the State Supervisor of Special Education. This committee should investigate the institution prior to commitment to determine if the school is properly equipped for education of the type of case referred to it. It is recommended that the parents of the hard-of-hearing child meet with this committee.

The State Supervisor of Special Education or State Social Worker is charged with the responsibility of executing the recommendations of this committee.

VII. Manual on the technic of testing the hearing of school children.

A Manual on the Technic of Testing the Hearing of School Children is in preparation by the members of your Academy's Committee on the Conservation of Hearing of School Children. This manual, among other things, stresses and includes the following:

1. An outline of the recommended statewide program.
2. Detailed procedures in the various recommended testing technics, including instructions on the selection of a quiet room for the testing and methods of checking the calibration and repair of testing instruments.
3. A model set of forms or blanks necessary for the administration of the entire program.
4. Sample forms of letters or pamphlets to teachers and parents on mental therapy and methods of assisting the hard-of-hearing child.
5. Description of a method of testing hearing which requires no equipment and which can be used by the average teacher or school nurse. This method will enable the examiner to detect hearing losses sufficiently severe to retard educational progress, but cannot detect incipient loss or high frequency loss in a quantitative manner.
6. A bibliography of pertinent references.

RESULTS OF IRRADIATION OF MALIGNANT MELANOMAS OF THE UVEAL TRACT

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THIS study has been undertaken in an effort to determine the effect of irradiation upon intra-ocular malignant melanomas. Our motive in pre-enucleation irradiation has not been to cure melanoma in any instance. We have sought to determine whether it is possible to "sterilize" the growth, so to speak, thus affording greater insurance against recurrence of metastases elsewhere in the system, or even against local recurrence. For this purpose all records and specimens in the ophthalmic laboratory of the University of Minnesota have been collected and analyzed. They include 113 proven melanoma cases, 79 of which were seen by one or both of us before enucleation. A further objective was to learn whether or not x-rays and gamma rays exert *any* favorable alteration in the morphology of the malignant growth. For this purpose sections of 28 eyes of patients treated by irradiation before enucleation have been compared with sections of 55 eyes enucleated from patients who did not receive such treatment after diagnosis. This histologic evidence will be illustrated later.

The statistical facts have been difficult to determine and are even now only offered as a preliminary report. We hope to have more complete data based on longer observation permitting analysis at a later date. Difficulties were encountered in obtaining complete medical histories of patients whose eyes were submitted for histologic study. The exact irradiation dosage was not definite in a few patients who were treated elsewhere. It was not always easy to secure accurate followup data concerning patients who changed addresses or who were dead. In six cases no record was obtainable. Fellow ophthalmologists and family physicians have been exceedingly cooperative in contributing facts regarding histories and specimens and in the followup of cases. Colonel J. E. Ash and Mrs. Helenore C. Wilder of the Army Medical Museum have generously aided in typing of tumors and reports of some of the specimens. Treatment was encouraged by the Tumor Clinic and Professor K. W. Stenstrom, Chief of the Department of Radio Therapy, of the University of Minnesota, in order to determine the actual facts regarding radiosensitivity of uveal malignant melanomas with special reference to types, under Callender's classification. To all these the authors wish to express their appreciation and gratitude.

The question might be raised whether or not the radioresistance of melanomas has been definitely established. Radioresistance of some sarcomas is generally acknowledged, but not very definitely as it pertains to malignant uveal melanomas. The bold statement has been accepted without very conclusive proof. Practically no evidence has accumulated in ophthalmic literature to condemn *prophylactic* radiation ther-

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apy for prevention of metastases from primary uveal malignant melanomas. Data have not accumulated regarding the possible difference in degree of resistance of the various *types* of malignant melanomas of the uveal tract. It was hoped that by accumulating a sufficient number of typed cases, some facts would be forthcoming, showing that certain types were less resistant than others. Our research has not proven this point. One might question the justification of pre-enucleation irradiation on the ground that valuable time was lost and that metastases, or extra-ocular extensions, *could* develop during the delay incident to irradiation and the advised waiting period before removal of the eye. On the other hand, one never knows the length of time a tumor has existed. In many cases the tumor mass was so large one might infer that it must have been quite old. Observation shows that some tumors grow slowly, others quite rapidly. Histories of visual impairment or of retinal detachment varied from a few weeks to several years. Doubtless in nearly every treated case there was plenty of time for metastasis or extension to develop before the melanoma was diagnosed. Unless it can be proved that irradiation aggravated the tumor growth, which is a mooted point, there was probably no harm done in the delay incident to attempt to "sterilize" the growth by irradiation before enucleation.

The prognosis for malignant melanomas in other parts of the body has always been considered very unfavorable regardless of treatment. Bloodgood[2] reported only one five year cure among over 200 malignant melanomas operated upon. Broders and MacCarty[3] traced only four patients with five year cures in 70 patients seen at the Mayo Clinic. On the other hand, patients with malignant melanomas of the choroid have enjoyed a relatively favorable prognosis. They are really not comparable with melanomatous cancers elsewhere in the body. Collins and Lawford[7] found 25 per cent of patients alive three years after enucleation. Von Hippel,[23] analyzing 189 cases of malignant melanoma of the choroid, found 65 patients alive after five years, 45 alive after 10 years, and 11 alive after 20 years. He states that 45 per cent of persons having melanomas die from metastasis. Our records reveal that nearly all these patients die from metastatic recurrence of malignancy. In the series of cases reported by Terry and Johns,[21] practically one-third developed metastasis within 12 years, the majority within five years. Metastases frequently occur within one or two years after enucleation, but the patient is never exempt from recurrence elsewhere in the system. In a case of Dr. Anderson Hilding's, (1922-599-N.A.K.) metastasis in the chest and local recurrence in the orbit developed 16 years after enucleation. Newton[14] reported a local recurrence in the orbit 13 years after enucleation, histologically identical with the primary intra-ocular growth, without any evidence of metastasis elsewhere, at that time, but he recently informed the authors that this patient subsequently died from metastasis. In Pahwa's series, 54 per cent were alive after five years. Thus one must conclude that, regardless of therapy or the stage when enucleation is done, the prognosis for malignant melanomas of the uveal tract is almost 50 per cent favorable but that it varies according to type. In our series of 65 patients whose eyes were enucleated prior to 1938, 37 (57%) were five year cures.

The greatest contribution to the subject of prognosis has come from the U. S. Army Medical Museum through the work of Callender, Ash and Wilder, in which work the American Academy of Ophthalmology and Otolaryngology has consistently maintained a special interest. Callender[5] demonstrated that study of the various cell types of uveal malig-

nant melanomas is of great prognostic value; that spindle cell types, especially "spindle A," metastasize almost not at all. His classification into spindle cells (A & B), fascicular, epithelioid and mixed cell types (with malignancy in the order named) and the Wilder[24] argyrophilic staining technic are of intense value in determining probability of malignancy and metastasis. Callender and Wilder[6] followed 120 cases of malignant melanomas and found no metastases had developed in these patients in which all areas of the specimens contained argyrophile fibers (Group III, on a basis of subgroups I, II, III). Unfortunately, preoperative biopsy studies are impracticable, but study of the sections affords very valuable aid in determining the prognosis after the malignant melanomatous eye has been enucleated. T. R. Pahwa[15] has contributed further evidence in support of Callender, Ash and Wilder's work on prognosis, based on the fiber content of the tumor.

The size or age of the growth affords no help in prognosis, and evidence of extension outside the globe does not necessarily indicate an early death. Even in very small tumors, we commonly found tumor cells present within the lumen of vessels in the tumor matrix, as noted by Byers and MacMillan[4]; extension along emissary vessels and nerves, the optic nerve or its sheath, was frequently noted. Penetration through the sclera with orbital involvement is not commonly observed. In our series it occurred five times. Even though serial sections were not studied, invasion of the scleral tunic was noted during microscopic study of sections, or found macroscopically, in 22 of our entire series. There was extensive orbital involvement in four cases when first seen, but in two of these orbital exenteration was not resorted to because of evidence of metastasis elsewhere. In seven instances observed, or in reported "followups," local recurrences developed. Aside from metastasis, two patients have died of other causes.

Although acute or absolute glaucoma, or increased tension, was recorded in 35 instances, there were 10 cases in which the tumor was not diagnosed until the eye was opened in the laboratory. An unsuspected tumor was revealed in two phthisical globes. One would expect cases which have reached the glaucoma stage to be more likely to develop metastasis or, at least local recurrence, but such is not the case. In only one of these glaucomatous eyes, many of which also showed evidences of tumor cell migration, has secondary orbital growth developed to date, whether radium was inserted or otherwise, but metastasis has developed elsewhere in 11 instances.

The notation of finding global extensions without development of secondary growth in the orbit seems inconsistent. It may be explained by atrophy or necrosis of tumor cells, by their phagocytic or chemical destruction locally, possibly in some instances by the frequent use of radium after enucleation. Following enucleations for preoperatively diagnosed malignant melanoma, 50 mg. of radium has usually been implanted in Tenon's capsule for from 12 to 20 hours. In several instances when radium was not available or when section of glaucomatous eyes revealed a growth, x-ray treatments were substituted. In only one of 49 cases receiving radium or x-ray after enucleation has there been a local recurrence. Without longer observation and further analysis of cases irradiated after enucleation, we are not yet prepared to assess its value in preventing metastasis. According to Duke-Elder,[8] "The value of radiation as a postoperative measure is undoubted; but as a primary method of treatment it is extremely precarious and in the present state of knowledge is unjustified. It has been very rarely attempted." ——"but, as a primary method of ther-

apy, any such attempts should be limited to cases wherein an only eye is affected or when other very special indications exist."

Incident to the origin of these tumors and their pigment content, Pohle[16] says, "Most of the neoplasms miscalled sarcomata are really melanoepitheliomata," and, "As in melanotic tumors generally, radiation therapy has comparatively little effect on such growths, which are highly malignant and resist irradiation." As a matter of fact, melanin of itself may have little relationship to malignancy. Melanin is not a part of the mesoblastic tissue and, according to Knight,[11] is produced only in ectodermal cells. Its occurrence in melanoma is perhaps incidental and no more frequent than it would be in melanoepithelioma. LaFerla[12] believes these tumors are all ectodermal in origin and that all so-called uveal melanotic sarcomata are really neuroepitheliomata. Moreover, Theobald,[22] Masson,[13] and others have produced quite conclusive evidence that "choroidal sarcomas" are not of mesoblastic origin but arise from cells from the sheath of Schwann, which is ectodermal tissue. Even though the above conclusions are correct, there is no definite evidence that tumors originating from ectodermal cells but involving mesoblastic tissues are less radioresistant than those of mesoblastic origin.

McKee, Coley and Huguet, Wood, and many others have condemned x-ray therapy, according to Ellis,[9] as not only valueless but as actually harmful. They even affirm that melanotic tumors are aggravated and metastasis is favored by its use.

Reese[17] made the terse statement, "Irradiation is of no value in any type of malignant melanoma of the uveal tract." Ellis[9] believed this attitude was not well founded. He reported a series of cases which presented a more optimistic viewpoint but finally concluded that general and local resistance was of greater and more significant importance. The chief proponents of radiation therapy have been radiologists, but they have not entirely based their evidence upon experience.

It was Sattler's[18] opinion that there was a more rapid and vast development of metastasis after intense irradiation of sarcomas. He did, however, state that irradiation performed in the "most practical way" might succeed in stopping further growth and even produce shrinking of the tumor, sometimes with all signs of necrosis; that there was no definite conclusion as to whether the inhibition of further growth would be permanent.

H. C. Stallard[20] made attempts to treat melanoma of the choroid by implants of radium and radium sewed to the sclera over the tumor. He concluded, "There is no doubt that for a time the neoplasm becomes more translucent and smaller. It is probable that some of the sarcoma cells are destroyed and that the tumor cells treated by phagocytes are eventually absorbed by the blood vessels." Stallard believed that the actual insertion of a radon seed is preferable to any other method of irradiation for intra-ocular neoplasm. That, however, is not often practicable because of the risk involved in damage to adjacent tissues. Even though the tumor, as in his cases, has shown definite regression, moderate necrosis and definite histologic changes in the cytology, practically all his cases came to enucleation. Berens[1] states, "It is too early and the clinical material is too limited to make any definite statements about the value of this form of therapy in intra-ocular sarcoma."

Because of several instances of rather rapidly developing metastasis following enucleation, one of us attempted, perhaps unjustifiably, to improve the prognosis of malignant melanomas by preoperative irradiation with gamma rays. The fact that many cases were seen after

the growth had evidently existed for many months supported the opinion that a delay of four weeks or thereabouts, when the full effect of irradiation could be expected, could probably afford only slight further opportunity for metastasis to develop. Moreover, several patients with small tumors were seen with excellent vision still maintained at the time of diagnosis. Some of these patients were not reconciled to enucleation. For these, and in one case with a melanoma of the choroid in the only eye with some vision remaining, and another in which the fellow eye was highly myopic, this treatment seemed rational.

Consultation with roentgentherapists resulted in the advice to employ crossfire irradiation anteriorly and laterally, with a total dosage of 1500 roentgens, as measured in air, to each field, divided over a period of 15 days; for the anterior field a 4x5 centimeter, and for the latter a 5x6 centimeter field. In the main, a 200 kilovolt, 60 centimeter target skin distance, and one-half millimeter copper filter for the anterior field and 1 millimeter copper filter for the lateral field was used. One millimeter of aluminum was always used as secondary filter. While this was a rather arbitrarily chosen method, and while perhaps a smaller dose given daily over a long period with a larger total dose might have been better, the procedure outlined above seemed best to the radiologists consulted. Whenever possible, at least one month elapsed after irradiation before enucleation was performed.

Thirty-six cases were irradiated, in most instances according to the above formula. Of these 36, only 21 were operated upon prior to 1938 and could therefore be considered in any analysis based on "five year cures." Analysis of the results in this small series cannot be conclusive nor has sufficient time elapsed to appraise fully the effects upon the development of metastasis.

The prognosis for melanomas of the uveal tract is dependent upon many factors, including general and local resistance of tissues, the length of time the tumor has existed, the cytology and reticulin content of the tumor, the thoroughness of removal, the trauma involved and the age of the patient.

Unless the factor of age is controlled, a larger proportion of "five year cures" in one group than in the other may result simply because the first group contains a larger proportion of young persons who naturally have a longer life expectancy. To check the possibility of

TABLE I.

Age of Irradiated and Non-irradiated Patients In Whom Enucleation Was Performed Prior to 1938.

AGE	IRRADIATED		NON-IRRADIATED	
	Number	Per cent	Number	Per cent
19	-	-	1	2
20-29	2	10	3	7 20
30-39	-	-	5	11
40-49	7	35	3	7
50-59	5	25	11	25
60-69	3	15	14	32
70-79	3	15	7	16
Age not reported	1		-	-
Total	21	100	44	100
Average age	52		55	

such a bias in the data, the age compositions of the two groups were compared and are shown in Table I.

Table I shows that the age distributions for the two groups differ considerably. The principal differences are (1) a considerably larger proportion of persons under 40 years of age in the non-irradiated group than among those irradiated (20 per cent and 10 per cent), (2) a much larger proportion of persons in the forties among irradiated persons than among the non-irradiated (35 per cent compared to 7 per cent), (3) a much larger proportion of persons in the sixties among the non-irradiated than among the irradiated (32 per cent as against 15 per cent). Despite these differences, the *average* ages for the two groups did not differ greatly, being 52 for irradiated cases and 55 for non-irradiated.

Because of these differences in age distribution and because of the obvious importance of age in determining longevity, it was felt necessary to control the age factor in Table II showing the number and proportion of "five year cures" in the two groups. This was done by reporting data separately for each of three major age groups, (1) patients under 40 years of age, (2) those 40 to 60, and (3) those 60 to 80. Although this procedure of classifying the cases into so many categories results in having too few cases of any one kind to provide definite proof of the effects of irradiation, it also prevents us from attributing to irradiation results which are heavily influenced by other factors, especially age and type of cell.

Table II shows that for the 21 irradiated patients operated upon before 1938, "five year cures" were obtained for 14, or two-thirds of the total. For non-irradiated patients operated upon prior to 1938 "five year cures" were obtained for only a little over half the cases (23 "cures" from a total of 44 enucleations). Irradiation seems to have been much more successful in the case of younger persons than for persons 60 years old and over. Of six persons of this older age group treated by irradiation, only one experienced a "five year cure," while a third of the 21 non-irradiated persons of these ages experienced such cures. Correspondingly, among the 14 persons under 60 who were given such treatment, all except two enjoyed "five year cures," while among persons under 60 who were not treated the proportion of cures was 89 per cent for persons under 40 and 57 per cent for those 40 to 60 years old.

If one considers age alone in computing five year cures, the summarized figures indicate 67 per cent as against 52 per cent in favor of irradiation before enucleation. Thus age influences statistics which may be entirely misleading if one fails to consider cytology and changes which should be readily recognized if irradiation exerts any favorable influence on the individual cell. However, this leaves out of all account the even greater importance of changes in cytology in the tumor submitted to irradiation.

In the pathological study of our tumor cases we have followed very closely the cell type classification of Callender and the fiber content classification of Callender and Wilder. Under this histological classification the greatest number of tumors in the here-reported series falls into Group II (Spindle B) and the mixed cell type V. The percentage of tumor cell types in this series of 113 cases corresponds closely to that reported by Callender. From a therapeutic standpoint the most radiosensitive groups should be Groups IV, III and V, i.e., epithelioid (IV), fascicular (III), and mixed cell type (V). These cell types contain wholly or in part epithelioid or young undifferentiated cells which should be "hard hit" with irradiation as compared to spindle cells, especially spindle cell subtype A (I) which

TABLE II.

"FIVE YEAR CURES" AMONG IRRADIATED AND NON-IRRADIATED PATIENTS.

Patients Operated Upon Prior to 1938, By Cell Type and By Age.

TYPE OF CELL	PATIENTS UNDER 40 YRS. OLD						PATIENTS 40-59 YRS. OLD						PATIENTS 60-79 YRS. OLD						TOTAL—ALL AGES					
	Irradiated			Non-Irradiated			Irradiated			Non-Irradiated			Irradiated			Non-Irradiated			Irradiated			Non-Irradiated		
	Total Enuc.	5 Yr. Cures		Total Enuc.	5 Yr. Cures		Total Enuc.	5 Yr. Cures		Total Enuc.	5 Yr. Cures		Total Enuc.	5 Yr. Cures		Total Enuc.	5 Yr. Cures		Total Enuc.	5 Yr. Cures		Total Enuc.	5 Yr. Cures	
		No.	%		No.	%		No.	%		No.	%		No.	%		No.	%		No.	%		No.	%
I	..	..	..	2	2	100	1	0	..	..	..	..	..	..	..	3	1	33	1	0	..	5	3	60
II	1	1	100	2	2	100	4	4	100	5	3	60	1	0	..	3	2	66	6	5	83	10	7	70
III	..	..	..	1	1	100	..	..	..	1	0	..	1	0	..	2	1	50	1	0	..	4	2	50
IV	1	1	100	1	0	..	1	0	..	3	3	100	1	1	100	8	1	11	3	2	66	12	4	33
V	..	..	..	1	1	100	4	4	100	2	1	50	3	0	..	1	0	..	7	4	57	4	2	50
Type unknown	..	..	..	2	2	100	2	2	100	3	1	33	..	..	..	4	2	50	3a	3a	100	9	5	55
Total all types	2	2	100	9	8	89	12	10	83	14	8	57	6	1	17	21	7	33	21	14	67	44	23	52

a—Includes 1 case for whom age is not reported.

has a very achromatic nucleus and seldom a nucleolus. Spindle subtype B (II) has a fairly large oval nucleus and large nucleolus; according to Broders, spindle cell subtype B (II) may be fairly malignant.

Systematic study of similar cell groups which have been subjected to pre-enucleation irradiation when compared with those which received no irradiation *shows practically no change in the microscopic pathology*. One is not able to demonstrate any destruction of cell nuclei, reduction of mitosis, increased necrosis of cells, or destruction of vascular supply. Sections of eyes which have been irradiated show no change in the relative extension of tumor cells along emanating vessels and nerves. A mixed series of specimens was submitted to other pathologists and they were asked to select the sections which showed results of irradiation. The invariable reply was that it could not be done. Some sections showed necrosis regardless of irradiation—no differential diagnosis.

Continuous followup of the complete series of individual cases reported in detail in Table III will provide a larger number of cases on which to base our conclusions and may require some changes in the findings shown by Table II.

We have had opportunity to observe, ophthalmoscopically, the results of x-rays and radium treatment on tumor growth in several instances. There was a definite regression in the size and appearance of the tumor after thorough cross-firing with x-rays in a few instances; the tumors continued to increase in size at a later period, in one case very definitely. In another patient there was observed a definite temporary shrinkage of the tumor to about two-thirds its original size six weeks after completion of irradiation. It is opportune to describe this case, observed over a period of 22½ years, in which the malignant melanoma definitely originated from a benign melanoma in the choroid, as suggested by Grinker.[10] The original choroidal "blue ointment" patch remained unchanged during a period of 12 years before becoming malignant. Enucleation in this case was deferred because it was the patient's only useful eye.

CASE REPORT

Mrs. Ella S., aged 46, was first seen at the University Eye Clinic in February, 1920. The right eye was amblyopic and divergent. Vision was 1/200, with a normal fundus. Vision of the left eye was 20/25 and J 0.50. In this eye a grayish "blue ointment" patch was noted above the left optic nerve and macula, two disk diameters in size, fairly sharp edged, not elevated. There was a positive scotoma corresponding to and extending vertically beyond the involved chorioretinal area. Since her husband had died of tuberculosis, she was thoroughly examined by the staff with negative findings throughout. Her Mantoux and Wassermann tests, including spinal fluid, were negative.

Under personal observation of one of us (F.E.B.) this fundus picture remained unchanged over a period of 12 years. A gradual decrease of vision to 20/50 and J 0.75 was recorded December 10, 1932, and she was more conscious of the scotoma. She stated that she noted a diminution in vision one month previously.

A definite elevation of this mass was noted with a slight retinal detachment above it, and a corresponding increase in the scotoma was recorded. Tumor was suspected, and two months later it became distinctly prominent, projecting forward above the macula into the vitreous with its rounded apex some 4 mm. above the level of the adjacent retina. There was no marked increase in the growth laterally. Since this was her only useful eye, on February 15, 1933, she was sent to the University Tumor Clinic for radiation therapy. Heavy irradiation by Professor Karl W. Stenstrom produced intense local reaction for some weeks. The tumor actually decreased in size during the following five months. A peculiar whitened area developed over its crest; there was no vascularity of its surface and its lateral dimensions seemed unchanged, but its elevation was noticeably less.

On July 27, 1933, vision was 10/200, the scotoma had extended laterally and vertically, although the slight detachment above the tu-

TABLE III.

TYPE I.

Spec. No.	Name	Age at 1st Exam.	Sex	Symptoms First Noted	Enucleation	Extraocular Extension	Glaucoma	Pre-op. X-ray	Post-op. X-ray	Living	Dead	Metastasis or Local Recurrence	Remarks
32-73	L. A.	61	F.	2½ yrs.	6-16-32		Absolute		X				
24-336	A. C.	74	F.	6 mos.	4-17-24		Absolute			No record since '26			Reported "ailing". Presumably dead.
32-59	J. E.	68	M.	1 yr.	5-26-32	Equatorial staphyloma			X	10-6-42			
42-177	V. F.	49	F.	3 yrs.	6-23-42			X		10-1-42			
22-559	N. A. K.	29	M.	6 mos.	5-16-22						8-13-41	Lung invaded in 6-7-39 and recurrence in orbit.	
30-92	A. L.	55	F.	6 mos.	7-11-30			X			May, 1933	Brain and vertebra.	Ext. along optic nerve.
31-14	Sr. M.	34	F.	12 mos.	1-6-31					8-10-42			
38-207	C. S.	82	F.	6 yrs.	8-17-38		X			8-13-42			

TYPE II.

Spec. No.	Name	Age at 1st Exam.	Sex	Symptoms First Noted	Enucleation	Extraocular Extension	Glaucoma	Pre-op. X-ray	Post-op. X-ray	Living	Dead	Metastasis or Local Recurrence	Remarks
39-144	M. B.	54	F.	1 mo.	8-7-39	N. R.		X	X	10-1-42			
M40959 M73669CR	M. C.	48	F.	1 mo.	5-29-40				X	9-1-42			
24-484	O. C.	51	M.	8 mos.	8-5-22	Orbit involved.	Absolute		X		Feb., 1925	Local rec. in orbit. Generalized sarcomatosis incl. brain.	
37-176	W. T. C.	71	M.	4 mos.	11-17-37						Feb., 1940	N. R.	Pigmented skin tumors.
41-187	M. D.	79	F.	6 yrs.	3-18-41		Absolute			9-22-42			
41-23	S. E.	22	M.	1 mo.	1-30-41		Acute		X	5-5-42			
42-1	A. J. F.	59	F.	1 yr.	12-31-41	Along scleral vessels and nerve.			X	9-12-42			
42-3	W. G.	56	M.	7 yrs.	1-7-42		Acute			9-9-42			
	M. L. G.	55	F.	8 mos.	5-18-31		Absolute	X	X	8-15-42			
36-126	W. K. J.	52	M.	5 yrs.	9-8-36				X	10-1-42			Diag. 18 mos. before enuc.
Bell	E. K.	75	F.	N. R.	6-27-39						8-1-42	Recurrence in orbit.	
27-1134 Bell	W. M.	55	M.	N. R.	July, 1927					1932			No record after 1932.
37-111	E. N.	36	M.	1 mo.	5-6-37	Along vessels and nerves.				9-1-42			
M24-1027	C. J. Oh.	27	F.	11 mos.	11-29-24		Acute	X	X		Oct., 1942		Sarcoma—liver.
40-108	C. O.	21	F.	10 yrs.	5-14-40			X		9-5-42			Melanoma disc. during pregnancy. Many pigmented moles on skin.

TYPE II. — Continued

Spec. No.	Name	Age at 1st Exam.	Sex	Symptoms First Noted	Enucleation	Extraocular Extension	Glaucoma	Pre-op. X-ray	Post-op. X-ray	Living	Dead	Metastasis or Local Recurrence	Remarks
40-193	J. O.	62	M.	7 wks.	9-18-40			•			9-14-41	Liver.	
31-22	M. P.	54	F.	1 yr.	4-7-31			X	X		10-2-42		Many pigmented moles on skin.
38-310	L. P.	28	M.	N. R.	11-17-38			X		6-20-40		N. R.	No subsequent record.
28-11	M. P.	65	F.	1 yr.	10-7-28	Yes	Secondary		X	8-10-42			
41-186	R. P.	35	F.	6 mos.	8-20-41					8-1-42			
39-37	D. R.	38	F.	3 mos.	3-12-39				X	9-28-42			
39-155	L. R.	41	M.	1½ yrs.	8-12-39		Acute	X	X	10-1-42			Refused enuc. 1½ yrs. until glaucoma.
22-190	O. R.	65	F.	3 mos.	5-3-22				X		11-12-27	Liver.	
40-122	E. S.	47	F.	3 mos.	5-27-40	O. N. invaded	X			9-4-42			Psychopathic.
34-88	E. S.	46	F.	2 yrs.	10-14-34		Secondary	X	X	10-1-42			Psychopathic. R. E. amblyopic. Benign mel. of chor. 3-24-20 from which tumor orig. Refused enuc. 2 yrs. until glauc.
36-62 (29-17)	I. S.	70	F.	3 yrs.	4-6-29		Absolute	X	X	N. R.	N. R.	N. R.	
37-117 SG065903	J. S.	41	F.	3 mos.	8-29-37			X	X	9-25-42			
38-144	W. A. S.	46	F.	4 yrs.	6-8-38			X		9-5-42			
	A. T.	39	M.	1 yr.	8-16-34	Sclera			X	9-10-42			
36-74 SG065900	A. W.	57	F.	4 yrs.	7-9-36	Yes	Acute		X	8-25-42			
30-85	C. W.	50	M.	1 yr.	12-6-30	Along perforating vessels.	Secondary				1-2-31	Liver.	

TYPE III.

Spec. No.	Name	Age at 1st Exam.	Sex	Symptoms First Noted	Enucleation	Extraocular Extension	Glaucoma	Pre-op. X-ray	Post-op. X-ray	Living	Dead	Metastasis or Local Recurrence	Remarks
41-103	A. B.	52	F.	3 wks.	4-3-41					8-15-42			Radon seeds sewed to sclera. Removed after 10 days.
42-176	D. D.B.	67	M.	1 yr.	9-2-42					10-1-42			
M5889	R. B.	57	F.	N. R.	10-5-22				X		Oct., 1926	Liver, June, 1926.	
40-105	E. D.	27	M.	4 mos.	5-6-40				X	9-10-42			
22-120	W. H.	39	M.	N. R.	2-6-22						4-10-35	Liver.	
34-105	G. J.	62	M.	2 yrs.	12-27-34	Sclera perforated.	X		X		June, '39 cause unknown	N. R.	History of trauma.
29-2	L. R.	62	F.	1 wk.	1-14-29						1934	Liver.	
42-09	R. S.	26	M.	6 mos.	10-10-40		Secondary	X	X		10-5-42	Liver, 9-28-42	
22-459 Bell	A. T.	63	F.	8 mos.	7-24-22		X	X			1924 cause unknown	N. R.	
42-155	R. S. T.	50	M.	10 days	8-11-42				X	10-1-42			

TYPE IV.

Spec. No.	Name	Age at 1st Exam.	Sex	Symptoms First Noted	Enucleation	Extraocular Extension	Glaucoma	Pre-op. X-ray	Post-op. X-ray	Living	Dead	Metastasis or Local Recurrence	Remarks
M424	G. A.	34	F.	6 mos.	5-23-24						8-17-25	Liver.	
30-54	M. C. (E.L.J.)	23	F.	2 mos.	11-20-30		Secondary				1940?	Liver.	No def. record after '40.
Mayo Cl.	F. S. E.	55	F.	3 mos.	3-15-41 Mayo Cl.				?	10-9-42			History of trauma.
34-12	E. H.	62	F.	5 mos.	2-9-34			X	X	10-1-42			
35-11	H. H.	64	F.	N. R.	1-28-35				X	N. R.	N. R.	N. R.	Cannot trace.
26-112	J. M. H.	66	M.	1 mo.	1-23-36						1-31-37	Orbital recur. Generalized melano-sarcomatosis.	Also leukemia.
37-102	O. H.	64	M.	2½ yrs.	5-29-37						Oct., '39	Liver & gen'l. sarcomatosis.	
25-153 Bell	M. H.	41	M.	5 mos.	2-20-25						Jan., '35	Liver.	
34-11	H. K.	64	F.	1 mo.	2-6-34		X			N. R.	N. R.	N. R.	Cannot trace.
39-154	G. L.	60	F.	6 mos.	Sept., '39			X		10-2-42			
39-34	L. L.	72	M.	N. R.	1936 M. G. H.		Acute		For orbit. recurrence		1-6-40	Orbital recurrence 7-15-38	Prev. oper. for detach.
22-462	N. M.	77	M.	2 yrs.	6-27-22 Exenteration	Orbital tumor	Secondary				1-5-23	Local recurrence liver and stomach.	Orbital extension.
40-69	A. N.	66	F.	1 yr.	4-9-40	X					Jan., '41	N. R.	Oper. for detach. 1938.
33-94	E. O.	63	F.	6 mos.	6-30-33					8-20-42			
34-95	S. O'R.	44	F.	1 mo.	11-22-34			X—	X		11-20-36		Shrinkage of tumor after irradiation.
33-105	G. R.	73	M.	1 yr.	9-26-33						10-17-35	Liver.	
39-183	M. R.	45	F.	Few days	10-17-39	In optic nerve.		X	X		12-15-41	Lung and liver.	
22-356 Bell	E. S.	48	M.	N. R.	4-12-22				X	9-24-42			
24-233	S. C. S.	N. R.	M.	N. R.	3-1-24					N. R.	N. R.	N. R.	Cannot trace.
25-948 Bell	Mrs. Z.	60	F.		9-16-25		Absolute			N. R.	N. R.	N. R.	Cannot trace.
37-119	E. Z.	42	F.	2 yrs.	9-2-37					9-20-42			

TYPE V.

Spec. No.	Name	Age at 1st Exam.	Sex	Symptoms First Noted	Enucleation	Extraocular Extension	Glaucoma	Pre-op. X-ray	Post-op. X-ray	Living	Dead	Metastasis or Local Recurrence	Remarks
41-110	C. A.	61	F.	3 mos.	5-2-41			X	X	8-22-42			
30-79 SG065897	C. A.	72	M.	6 mos.	6-19-30			X			Oct., '34	Liver.	
39-211	H. A.	20	M.	3 yrs.	11-21-39	Sclera perforated.				8-25-42			History of injury. 10 yrs. prev.
34-24	I. B.	52	M.	3 mos.	3-19-34				X		June, '36	General sarcomatosis.	
33-95	R. B.	48	M.	3 mos.	7-20-33			X	X	8-7-42			Amblyopic eye.
41-50	R. B.	64	F.	1 yr.	3-12-41			X		9-6-42			Ciliary body invaded
	C. DeZ.	58	F.	7 wks.	12-26-35					8-31-42			Verhoeff specimen.
31-6 SG065898	A. F.	72	F.	2 wks.	6-10-30	Along penetrating vessels.		X	X		Mar., '32	Liver.	
32-79	M. F.	52	F.	2½ yrs.	7-5-32	Through sclera.		X	X	9-20-42			
42-76	C. H.	45	F.	6 mos.	5-4-42					10-1-42			
36-125	K. G.	45	F.	1 mo.	12-16-36			X		5-18-42			
41-91	M. G.	65	F.	9 mos.	4-17-41		Secondary			9-8-42			Annular melan. of iris.
40-1	J. H.	47	M.	2 yrs.	12-28-39	Along scleral vessels.	X	X	X	8-24-42			

TYPE V. — (Continued)

Spec. No.	Name	Age at 1st Exam.	Sex	Symptoms First Noted	Enucleation	Extraocular Extension	Glaucoma	Pre-op. X-ray	Post-op. X-ray	Living	Dead	Metastasis or Local Recurrence	Remarks
24-1026 Bell	W. H.	19	M.	1½ yrs.	12-13-24		X			10-8-42			Reported A. J. O. 1926—Murray.
41-3	C. I.	66	F.	2 yrs.	12-31-26		Iridectomy for inc. tension		X		7-3-27	Lung.	Tumor discovered after glauc. & cat. oper.
39-79	C. O. J.	48	M.	6 wks.	4-21-39					10-1-42			
41-83	J. J.	66	M.	9 mos.	4-3-41					9-3-42			Iridocyclitis. Tumor undiagnosed postoper.
38-279	O. J.	45	M.	10 mos.	10-2-38					9-2-42			
37-Jan.	A. K.	60	F.	6 mos.	1-8-37			X	X		3-19-41	Abdominal.	
40-246	H. K.	54	M.	1 wk.	12-10-40		Absolute			9-18-42			
?	R. K.	40	M.	6 wks.	8-24-36			X	X	9-2-42			
38-183	A. N. L.	48	M.	5 yrs.	7-19-38	Orbital extension.	X		X		8-8-42	Liver, June, 1942	Glauc. oper. 1934 Recurrence 1937.
39-26	J. L.	50	F.	2 mos.	3-3-39	Along vessels.		X	X	8-25-42			
39-58 M39640	G. McI.	49	F.	8 yrs.	4-22-39	Along optic nerve.			X	8-15-42			Phthisis bulbi.
41-3	G. S.	62	M.	3 wks.	1-8-41	Optic nerve involved.		X	X		Jan., '42	Liver and chest.	
39-127	J. Y.	68	M.	1 yr.	7-18-39		Acute	X	X		Oct., '41	Liver, mediastinum & lung.	Refused enuc. 1 yr. until glaucoma.

SPECIMENS SPOILED, NECROTIC AND UNIDENTIFIED

Spec. No.	Name	Age at 1st Exam.	Sex	Symptoms First Noted	Enucleation	Extraocular Extension	Glaucoma	Pre-op. X-ray	Post-op. X-ray	Living	Dead	Metastasis or Local Recurrence	Remarks
	A. P. B.	N. R.	F.	3 mos.	10-25-35			X		9-25-42			Specimen untraced.
M9688	C. B.	74	M.	2 yrs.	11-22-23		Absolute				2-21-31 Heart.		
35-73	C. C.	54	F.	4 mos.	11-5-35				X		7-12-39	General sarcomatosis.	Subsequently in Class V. Spindle cell B epithelioid.
M26-425	L. G.	26	F.	3 yrs.	7-26-26				X	9-1-42			
St. L. N.O.	K. E. H.	55	F.	2 wks.	Mar., 1918					9-18-42			
26-120	G. C. H.	61	M.	1 mo.	12-20-26				X		Oct., '32	Liver, 8-16-32.	
M?	M. H.	78	F.	1 yr.	9-5-24	Orbit			X		Sept., '26	Liver.	
?	B. J.	47	F.	4 mos.	5-21-36			X		9-17-42			Cannot classify. Spec. dried up.
St. L. 1919 N. O.	J. K.	56	M.	2 mos.	5-31-19						11-29-23	Liver. Recurr. in orbit.	
	O. L.	69	M.	3 yrs.	5-21-26		Absolute		X		10-12-27 Pneumonia		
M?	D. O.	58	F.	2 mos.	7-17-36			X	X	10-1-42			
41-145	M. P.	47	F.	13 mos.	6-13-41	Sclera perforated.			X	8-11-42			History of trauma.
25-98	L. R.	20	M.	1 yr.	1-27-25		Absolute		X	9-11-42			
39-213	A. S.	80	F.		10-10-39			X	X	9-28-42			
							ENUCLEATION REFUSED						
	A. J.	58	F.		Diagnosis,	Dec., 1938.		X			12-11-41	Abdominal sarcomatosis.	Tr. by chiropractor.
37-137	M. S.	77	F.	3 yrs.	Enuc. not done because of massive ext.		X	X			June, '39	Liver.	Biopsy type IV. Melanoma. Lived 5 yrs. after 1st sym.
	S. W.	68	M.	2 yrs.	Refused	5-8-37.		X			9-1-42	General sarcomatosis	

mor mass was unchanged. Another course of x-ray treatment was given.

On December 11, 1933, vision in the left eye had improved to 20/200 and although the tumor projected farther forward into the vitreous, much like a pear, it had not grown laterally.

By March 12, 1934, the tumor had not extended, although the eye was "irritable" and pressure was 33 mm. Hg. (Schiotz). Pilocarpine was prescribed.

On July 19, 1934, vision was 20/65, pressure was 34 mm. Hg. (Schiotz) and the tumor was unchanged.

On August 20, 1934, following a coughing spell two days previously, when she described her vision as "blurred and cobwebby," the retina was found detached downward, the vision was reduced to counting fingers at one foot and the pressure was 35 mm. Hg. (Schiotz). Fundus details were not possible.

On October 19, 1934, the patient consented to enucleation when convinced that useful vision was lost and because the eye was painful (pressure 55 mm. Hg. Schiotz-Gradle), and 50 mg. of radium were inserted for 13 hours.

Examination of the eye showed a mushroom growth 8 mm.x 7 mm. x 7 mm. protruding from above the nerve and macula, the site of the original area of discoloration. Sections showed a spindle and small epithelioid cytology, moderately pigmented and cystic.

This patient is still alive, quite psychopathic, but with no evidence of metastasis 10 years after diagnosis was established.

There was no doubt from ophthalmoscopic observation over a period of 21 months that in this case tumor growth was definitely arrested for a time at least. It was an ideal case for clinical study of the effect of irradiation owing to the refusal of this patient to part with her only seeing eye until glaucoma developed and vision was lost.

Study of sections of irradiated eyes affords the most definite evidence of the effect of irradiation upon the tumor cells. In fact, it is the only conclusive evidence available. C. M. Scott[19] believed the effect is greatest in the dividing nucleus. Since tumor cells multiply more rapidly than normal cells and, being younger cells, should be more readily damaged than normal cells, one naturally infers that the biological effects of irradiation, in proper dosage, should be more effective against rapidly dividing malignant cells than it is against neighboring normal cells. But what is "proper dosage"? There is no standard or fixed melanoma dosage. Some cell types are more radioresistant than others. Exact dosage cannot be predetermined. One might expect unfavorable sequences if normal adjacent cell resistance is damaged by improper quantitative irradiation, especially if local blood supply is impaired, but apparently clinical observation does not prove such to be the case. Necrosis of orbital tissue, marked vascular destruction and shrinkage of the orbit followed too intensive irradiation in some cases; in others, using the same dosage and screening, local reactions were not very marked or were fairly absent. One must therefore infer that "radiosensitivity" varies greatly, even when dealing with normal tissues, with all the improvement in radiation technic, measurement and distribution of dosage and more accurate filtration, safe dosage for intra-ocular therapy remains a question to be determined. It has never been accurately based upon histologic evidence. One cannot know "a priori" just what type of cells prevail in any given tumor; nor may one conclude that a rapidly growing tumor may possess greater or less radioresistance than one in which growth is retarded. Moreover, if tumor cells are radiosensitive, there should be a cessation of nuclear segmentation with diminished mitosis and evidence of granular and vacuolar degeneration of cell protoplasm. There should be an invasion of leucocytes to carry off necrosed cancer cells. Ultimately there should be some replacement by connective tissue.

As already stated, occasional regression of tumor growth was noted ophthalmoscopically. Microscopic search through hundreds of sections of irradi-

ated tumor-containing eyes reveals faint evidence of the aforementioned changes which should occur and which we had hoped would occur. Because changes sometimes occurred all too readily in orbital tissues outside the globe, with resulting contracted sockets which made prosthesis unsatisfactory, one might infer that the scleral tunic may act as a filter which prevents radio penetration and similar changes in the malignant melanoma.

From our experience with radium therapy in malignant melanoma for the purpose of arresting growth, preventing metastasis, or prolonging life, *the conclusion is definite that this form of therapy is valueless.* Whether or not irradiation immediately after enucleation when given for the purpose of preventing local recurrence or extension of tumor cells into the circulation accomplishes any purpose must for the moment remain *sub judice.* Longer study of this series may prove its value.

CONCLUSIONS

As a result of experience with a small group of cases, we must conclude that:

1. Even with intensive irradiation its action is valueless in any of the types of malignant melanoma.

2. Roentgen therapy offers no assurance against metastasis; there is even a possibility of proof, when complete figures are available for a longer series and covering a longer period, that metastases develop earlier after irradiation than when it is not employed.

3. Irradiation should be reserved for cases in which the tumor-containing eye is the only one retaining vision, for cases where the patient refuses enucleation and those in which the tumor has already invaded the orbit and adjacent structures.

4. Early diagnosis and immediate enucleaton remain of greatest value in prognosis.

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DISCUSSION

A. B. Reese, M.D., New York, N. Y.: The authors speak of the poor prognosis of malignant melanomas elsewhere over the body compared to those in the eye. They cited Bloodgood's figures of only one-half of one per cent cure in 200 cases of malignant melanomas elsewhere compared to the approximately 50 per cent cure of intra-ocular malignant melanomas. This discrepancy is not due to the difference in location of the same tumor, but to the fact that there are two different tumors called by the same name. Malignant melanomas elsewhere over the body are not only different from intra-ocular melanomas in regard to prognosis, but also in regard to their clinical and histologic characteristics. The one seems to be neurogenic in origin and the other ectodermal.

It seems to me the only factor common to these two tumors is the occurrence of pigment. Thirty-nine of the authors' cases had glaucoma, and in 12 of these the diagnosis of malignant melanoma was not made until the globes were opened in the laboratory, two with phthisical globes. This is not an uncommon experience. It emphasizes, therefore, all the more the possibility that an old glaucomatous eye may harbor a malignant melanoma and serves as another reason why patients should have such eyes enucleated.

Sometimes statements are perpetuated in the literature without any proven scientific basis or without sufficient data. Such has been the case in regard to the effects of irradiation on intra-ocular malignant melanomas. Doctors Burch and Camp now give us accurate information which indicates that irradiation of such tumors is without benefit in regard to arresting the growth, preventing metastasis or prolonging life. They admit that a longer period of observation would be advisable. Although the authors' findings are negative, it is important to have them established in the literature on a sound basis.

ANGIOMATOSIS RETINAE

A Report of the Successful Treatment of One Case

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ANGIOMATOSIS retinae is a relatively rare condition. Many experienced ophthalmologists have never seen a case. Bedell[1] in 1931 found that less than 100 cases had been reported. Cordes and Hogan[3] in 1940 found that the number in the literature had risen to 135. Since that time about ten additional cases have been reported.[4,8,13] The first description of the condition was by Fuchs[6] in 1882, who considered it an arteriovenous aneurysm. Wood[18] in 1892 and Collins[2] in 1894 described the next cases. Von Hippel[15] was apparently the first to realize that the disease was a clinical entity and since his publication in 1904 the condition has been called von Hippel's disease. In 1927 Lindau[9] reported that angiomatous tumors of the retina were often part of a general disorder characterized by angiomatous cysts of the cerebellum, medulla, spinal cord, suprarenals, kidneys, liver, epididymis and ovaries. Thus Lindau's disease became established as an entity. His viewpoint has been adequately substantiated by other observers. He considered that about 25 per cent of patients with retinal angiomatosis had intracranial complications, principally angiomatous cysts of the cerebellum.

As the literature on this subject has been adequately reviewed by others,[1,3,10] only a brief description of the disease will be given with the report of a case, treated a year ago by diathermic punctures. It is hoped that this presentation will prove to be of some value to those who are not thoroughly familiar with this interesting disease.

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OPHTHALMOSCOPIC FINDINGS

When an observer has once examined a typical case, or seen colored fundus photographs of the condition, recognition is fairly easy provided the characteristic appearance is present. However, the occurrence of bizarre types is not uncommon and may make the diagnosis very difficult. Duke-Elder[5] states that the usual clinical course may be divided into four stages: (1) the early stage of vascular dilatation and angioma formation; (2) the appearance of hemorrhages and exudates; (3) the stage of massive exudation and retinal detachment and (4) the final stage of secondary glaucoma and destruction of the eye.

All observers seem to agree that the first sign is an enlargement of a retinal vein which rapidly becomes greatly dilated and tortuous. The accompanying artery also enlarges. Shortly thereafter a rounded, elevated, reddish or grayish mass is found far out in the periphery of the fundus, especially on the temporal side. As the enlargement of the vessels continues, it may become impossible to distinguish the artery from the vein. While the angioma and the vessels are increasing in size, retinal edema and exudative changes become manifest. A star shaped figure may develop in the macula or an appearance similar to a circinate retinitis may be found. As Bedell[1] pointed out, the exudates, which at first consist of yellowish white flecks, by a process of

coalescence become subretinal sheets. Retinal detachments may precede these exudative changes, or may occur simultaneously. Eventually, complete retinal separation takes place and as Lloyd[10] has shown the diagnosis may be very difficult, unless an angiomatous mass and the enlarged vessels can be seen. Secondary glaucoma develops and enucleation of the eye usually becomes necessary.

DIFFERENTIAL DIAGNOSIS

When a case of retinal angiomatosis follows the usual pattern the diagnosis is relatively easy, but atypical or far advanced cases may be very difficult. Confusion with glioma of the retina has often occurred. Another condition which at times simulates angiomatosis is Coats' disease. Arteriovenous retinal aneurysms, choroidal tuberculosis, endophthalmitis and several other conditions such as brain tumor and kidney disease may occasionally be mistaken for angiomatosis. Careful and repeated ophthalmoscopic examinations, transillumination and slit lamp studies, combined with a proper evaluation of the history and physical examination, will usually lead to a correct diagnosis.

PROGNOSIS

While some cases may remain latent indefinitely, retinal angiomatosis is essentially a progressive disease. The prognosis therefore is definitely bad. Detachment of the retina or extensive exudation and hemorrhage leads to blindness, and secondary glaucoma leads on to enucleation of the eyeball. Thus all cases which are seen at an early stage should be given the benefit of some form of treatment because, otherwise, destruction of the eyeball is usually inevitable. If cerebellar symptoms are present the case should be referred immediately to a competent neurosurgeon.

TREATMENT

Before local treatment is begun it is wise to have a thorough general and neurological examination to determine if the case is simply one of von Hippel's disease or if it is Lindau's disease. If Lindau's disease, the cerebellar tumors should be attacked first and if successful, therapy of the eye should follow. Treatment may be attempted with roentgen rays, or radium, even in advanced cases, and although vision may have already been destroyed, irradiation may prevent the necessity of enucleation.

In early cases various attempts have been made to destroy the growth. Radium applications were employed by Holm,[7] Traquair,[14] Foster Moore[11] and others. Cordes and Hogan[3] reported definite improvement in one patient on whom they used high voltage roentgen therapy. Neame[12] treated a case by electrolysis. Weve[17] used surface coagulation with diathermy and also diathermic punctures. It was the latter method which was employed in the case reported in this paper. It is particularly applicable in cases with a single angioma located far in the periphery. It is true that damage to the vision may be extensive but not much more than that caused in the operation for cure of retinal detachment. Furthermore, if the growth is not destroyed in some manner, it will destroy the eye. As a rule, angiomas are located far in the periphery of the retina and are therefore readily accessible for treatment with diathermy. The ophthalmic surgeon may employ the same methods of localization of the growth as he is accustomed to use in localizing tears in cases of retinal detachment. The operative approach and technic are practically identical to that employed in the surgical cure of detachment of the retina by means of diathermy. Kaye[8] has reported two cases, a brother and a sister, whom Stallard treated with apparently perfect re-

sults by a combination of katholysis with surface and puncture diathermy.

TECHNIC

After localization of the angioma, that area of the sclera should be adequately exposed by a generous incision through the conjunctiva along the ora serrata. It will usually be necessary to draw aside a rectus muscle or divide the tendon and retract the muscle out of the field until the attack on the angioma has been completed. Surface coagulation may be employed over the area of the angioma and the surrounding retinal detachment. Scleral punctures with the Walker micropins above and below the growth should be made and observed with the ophthalmoscope to check the localization of the angioma. The formation of a bubble or what Walker[16] calls the "bubble vacuole" may be seen with the ophthalmoscope at the site of the puncture. Two or more punctures should be made just posterior to the angioma, either into or as near as possible to the enormously dilated vein and artery. After this, punctures directly into the angioma and the surrounding area of retinal detachment are made. The micropins should be inserted through the sclera about two millimeters apart. From 30 to 50 milliamperes of current are used. The divided rectus muscle is carefully sutured back to its stump with 0000 chromic catgut. The conjunctiva is then closed with a running silk suture. The postoperative care of the patient is similar to that of cases of retinal detachment except that it need not be as strict or carried out for as long a period.

CASE REPORT

History—W.K.B., a white male, 25 years old, was seen in October, 1941, because of failing vision of his left eye for the past six weeks. Recently there had been headaches after reading and also after working at his occupation as a cotton classer. There was no history of previous ocular troubles, headaches or dizziness.

The past history was without interest as was that of his family. His father and mother were aged 57 and 52 respectively and were in excellent health. The patient was an only child. He was married, but had no children. The parents' fundi were examined and found to be normal.

Ophthalmic examination—Both eyes were normal externally. Vision in the right eye was 20/25, corrected to 20/20 by a plus .50 cylinder, axis 90. Vision in the left eye was 8/200, unimproved by lenses. The pupils were normal and dilated fully and evenly under homatropine. The media of both eyes were clear, and the right fundus was normal.

The left fundus showed a most interesting picture. The disk was normal except for a marked dilatation of the inferior temporal vein. The accompanying artery was not enlarged until it reached a distance of two disk diameters from the papilla where it enlarged rapidly so that it became indistinguishable from the vein, except by its course. The enlarged vessels led to a tumor mass almost one disk diameter in size, near the ora serrata in the "3:30" meridian. The mass was nearly round, definitely elevated with a smooth surface and grayish color. In and around the macula were bright yellowish spots of "exudate," star-shaped above the macula and in a large sheet below. The appearance of the exudate was somewhat similar to that of circinate retinitis.

Observations of the fundus were made several times over a period of about three weeks, during which time consultations were held with Doctors E. C. Ellett and Ralph O. Rychener. It became apparent that the condition was a progressive one and that unless something were done the eye would have to be enucleated. It was decided to try to

destroy the growth and the dilated vessels by puncture diathermy. A careful physical examination revealed no abnormalities other than those of the eye.

Operation—On November 7, 1941, the operation was performed with the help of Dr. Rychener. Preoperative sedation and local anesthesia were employed. The area was exposed just as is done for a retinal detachment. The external rectus tendon was cut near its insertion and retracted to give adequate exposure. The pupil had been widely dilated to facilitate ophthalmoscopic examination during the operation. One Walker micropin was placed just above and one just below the growth, using a diathermy machine set to deliver 45 ma. of current. Micropins were then inserted posteriorly to the growth in an effort to strike or destroy the large feeding vessels. The vein was luckily transfixed with one pin, following which several were placed so as to thrombose both the vein and the artery. The "bubbles" produced by these punctures were observed with an ophthalmoscope to check the accuracy of the placements. Following this, the entire area of the tumor and that of the surrounding retinal detachment were studded with micropins. No hemorrhage occurred, so that ophthalmoscopic observation was possible throughout the operation. The external rectus was reattached with fine chromic catgut, and the conjunctiva closed with a running silk suture.

Postoperative course—The postoperative care was similar to that used after operation for retinal detachments. Both eyes were kept covered for seven days, after which pinhole goggles were used. The operated eye was kept atropinized for about six weeks. The patient was allowed to resume his work four weeks after operation. Colored fundus photographs were taken 12 days after operation and at various intervals since that time. These were shown to demonstrate: (1) the early changes consisting of retinal edema, hemorrhages and thrombosis of the enlarged blood vessels and (2) the progressive changes which have gradually led to the present condition of pigmented scar tissue. No enlarged vessels remain, but a few yellow spots persist in and around the macula. No improvement in vision occurred after operation, but the destruction of the growth and the saving of the eyeball with normal appearance and normal tension constitute at least a partial victory. Had the case been seen before extensive degeneration of the macula had occurred, better function probably would have resulted. A year has passed and no other angiomas and no cerebellar symptoms have developed.

SUMMARY AND CONCLUSIONS

1. Angiomas of the retina are discussed briefly regarding their symptoms, diagnosis, course, prognosis and treatment.

2. A rather simple method is described of treating these growths by puncture diathermy. It is especially applicable in cases of simple angiomas which are discovered early.

3. A case treated by this method and followed for one year is reported. Fundus photographs were exhibited, showing the condition before and at various stages since operation.

The writer wishes to acknowledge with thanks his indebtedness to Dr. Ralph O. Rychener for his many helpful suggestions and for his expert assistance in the management of this case.

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DISCUSSION

LAWRENCE T. POST, M.D., St. Louis, Missouri: The author is to be congratulated on the successful treatment of the local manifestation in the eye of this rare disease. Though the destruction of one lesion in this disease in which there may be multiple lesions may only postpone the evil day, as the author has pointed out, it is to be hailed as a great achievement and might, in more favorable cases, prolong useful vision for many years.

The author's point that the addition to the literature of new cases of this rare disease is desirable is well taken. It requires a long time for the clinical picture of unusual conditions to become so familiar to the entire profession that they will be on the alert to recognize them when encountered. Probably this disease is not so rare as has been thought. I am presenting three cases today, two of which I am sure have not been described in the literature, and Doctor Boots, past resident at Washington University, has seen three other cases in the examination of draftees at Jefferson Barracks in the first six months of his service. Photographs of one of Doctor Boots' cases are to be shown by me today. It is obvious that when a disease has once been seen and recognized new cases of a similar nature are much more likely to be identified.

The first case that I present is that of a 30 year old, married female, E. C. R., of Dallas, Texas, referred by Dr. J. Guy Jones, of Dallas, on April 19, 1937. She gave a history of dimmed vision in the right eye of four months' duration. Vision was O. D. 20/24; O. S. 20/15.

Just temporal to the right macula there was an area of healed inflammation or hemorrhage one disk diameter horizontally by one-half disk diameter vertically, over which the retina was somewhat elevated. The descending temporal vein was dilated to about twice its normal size. The accompanying artery was beaded for four disk diameters below the disk and then showed aneurysmal dilatation to about twice its previous size. It could not be differentiated from the vein. The artery and the vein terminated in a silvery grey mass elevated five or six diopters. At the apex of the mass was an orange ball, into which the artery and vein led directly. The top of the mass measured six diopters. The disk was somewhat blurred and showed hypergliosis. The cup was filled with new connective tissue and all veins were dilated. There were a few hyaloid areas in the lower periphery. Examination of the left eye revealed a normal macula.

Complete examination was not possible as the patient was making hurried rounds of the country to try to find someone who could help her. The correct diagnosis of the condition had been made by the referring ophthalmologist. The patient brought excellent illustrations of her fundi, both of which were extensively involved with characteristic vascular lesions and multiple "bees' nest" tumors, and the retina of the right eye was widely detached. I advised her that radium or deep x-ray treatment was the best therapy that I knew. Her next stop was to be in Kansas City where she was to see Doctor Curran. Whether her case has been recorded previously I do

not know. An interesting feature was that she had a brother — whom I did not see — similarly affected. His case was described in a letter to me by Dr. J. Guy Jones. Their father had one blind eye with white pupil. I wrote to her a month ago to find out what had transpired since I saw her, but the letter was returned unclaimed.

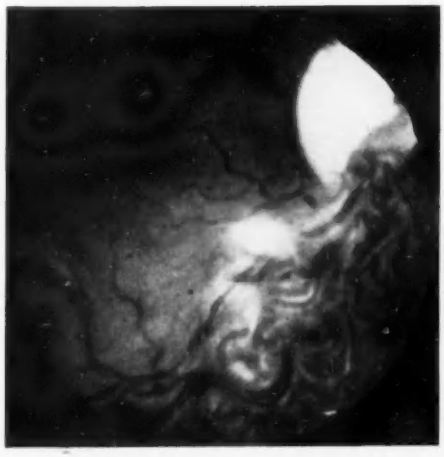

FIG. 1. Case 2 — Dilated vessels.

My second case was that of E. D., referred by Doctor Boots from the St. Louis induction center. I show a fundus photograph (Figure 1) of the patient's one involved eye. The greatly enlarged artery and its accompanying giant vein can be well seen. In this man's case there were no tumors visible and no evidences of cerebellar or other involvement, hence the diagnosis is presumptive. No treatment was given.

The third case was that of a 27 year old, single female, B. K., whom I first saw on May 16, 1942. She complained of decreasing vision in her better eye over a period of two weeks. Her other eye had always been defective, and on examination proved to be moderately amblyopic with vision of 20/50 and relative central scotoma. This fact naturally was an ever present threat in any contemplated therapy.

The vision of the left eye which contained the angiomatous tumor was 20/30. Figure 2 is a composite of two fundus photographs and illustrates clearly the two very large vessels leading from the disk to the tumor. Figure 3 shows more of the tumor. Obviously, this lesion is all in the extreme posterior pole of the eye near disk and macula, which fact also influenced therapeutic considerations. Edema and exudate had already extended into the macula causing the blurred vision of which the patient complained.

Three methods of treatment seemed worthy of consideration: Radium seeds, diathermy puncture and thermophore application. They

FIG. 2. Case 3 — Dilated artery and vein and part of tumor.

were preferred in the order named, chiefly because of the inaccessibility of the tumor. An earnest wish not to damage the macula further and not to rupture a large vessel in this, the patient's only eye, was never out of the surgeon's mind.

There was only one week in which the patient would be available for treatment because of her personal affairs. Radium seeds were ordered from New York. As ill luck would have it, they did not arrive as planned.

Preparations were made therefore to use diathermy, employing the convenient long and curved Gradle needle devised for retinal detachment operation. Again bad luck intervened and this needle could not be found. Hence, the method finally used was the thermophore. The awkwardness of the approach through the necessarily inadequate exposure was the real detriment to the operation. Figure 4 shows the tip employed. I devised this for treatment of retinal detachments some years ago. A glance will show that the elbow at the turn toward the point and the mass of the shaft, necessary to con-

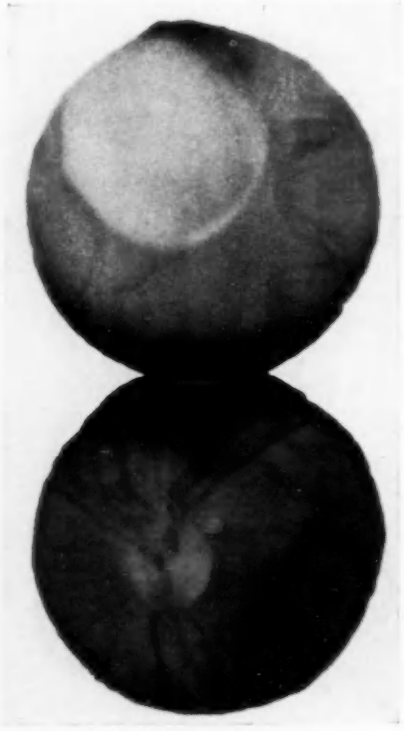

FIG. 3. Case 3 — The entire tumor.

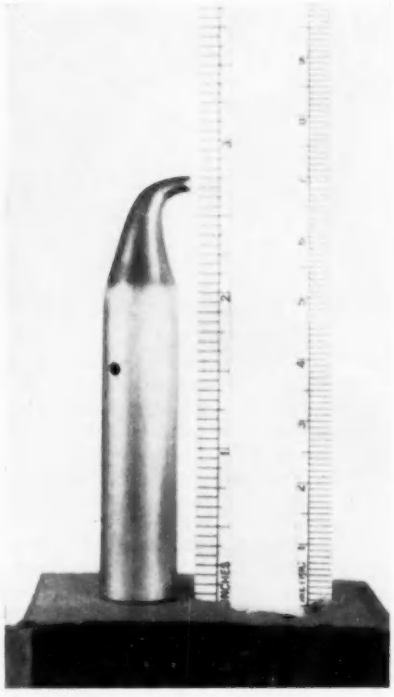

FIG. 4. Case 3 — Thermophore point used in treatment.

vey adequate heat to the tip, were too large for ready accessibility to the sclera opposite the growth. The eye had to be rotated downward so that direct vision during application was not possible, nor was it possible to observe the effect immediately so as to try again if the application had not been made at the desired point, because heating at 160°, which will destroy choroid and was thought would destroy the tumor, does not produce any immediately observable effect. After a prolonged struggle the point was placed over an area thought to be correct though the attempted scarification of the localized spot had not been satisfactory. The optic nerve could easily be felt and it was known that the tumor was approximately 4 mm. almost directly above it. What was not realized was the possible rotation of the ball in fixation of the globe. I was chagrined to find on examination of the fundus a few days later, when the great reaction had subsided, that the perfectly round white area corresponding to the thermophore contact surface was clearly visible just below and temporal to the macula! No opportunity for another picture to show this offered and when next seen this white area had entirely disappeared and the condition of the eye seemed the same as before the operation.

Encouraged by Doctor Lewis' success I may try again, using diathermy, though I hesitate to risk further damage to the macula which is so near the tumor.

JACK S. GUYTON, M.D., Baltimore, Md.: I wish briefly to present two cases in support of Doctor Lewis' conclusions.

The first case is that of a 13 year old girl with bilateral angiomatosis retinae. The patient has been followed by Dr. F. H. McGovern, of Danville, Virginia, during the past year. Under his direction her eyes were first treated with x-ray therapy. This failed to check progression of the lesions and during June, 1942, he referred her for diathermy operations.

I will confine myself to the result of operation on the right eye. The corrected vision in the eye prior to operation was 20/15 -2. The single angioma was localized by shining an ophthalmoscope on the lesion and marking the point where the light penetrated the sclera. Sixteen Walker pins were inserted, as in a detachment operation, into and just surrounding the tumor mass, their position being confirmed by frequent ophthalmoscopic examinations. A current of approximately 15 milliamperes for three seconds was used for each pin. A small hemorrhage appeared within the tumor during this procedure. No attempt was made to close off the nutrient vessels, and at the close of operation the vessels appeared almost as large as before, although the angiomatous mass had been completely obliterated.

Postoperatively the fundus showed a marked reaction. There was a moderate sized hemorrhage surrounding the obliterated angioma, and in the overlying vitreous a good deal of subretinal serous exudate was present producing a flat detachment of the entire upper half of the retina and some subretinal hemorrhage around the temporal side of the disk. This reaction slowly but completely subsided. Three weeks after operation the corrected vision was 20/50 -1. The retinal vessels gradually reduced to normal size within the first week after operation. Six weeks after operation all hemorrhage and exudate had disappeared, the site of the angioma was a healed scar and corrected vision had returned to 20/15 -2.

The second case is that of a seven year old girl with bilateral juvenile Coats' disease. This patient developed progressive failure of vision in her left eye during the summer of 1941. She was first examined in September, 1941, at which time the left eye was completely blind, with an advanced exudative retinitis. This eye was eventually enucleated, after a short episode of secondary glaucoma. Histological examination showed in the periphery of the retina on one side some unusually large blood vessels associated with an area of massive glial proliferation, and in places a large number of capillaries. There was massive subretinal exudation. There were newly formed vessels on the anterior surface of the iris and ectropion uveae. Although this histological picture was that of massive exudative retinitis and glial proliferation (Coats' disease) it suggested atypical angiomatosis retinae.

In September, 1941, the vision in the right eye was 20/20, and the eye was entirely normal with the exception of the fundus. The disk was slightly grayish in color and definitely blurred. In the far temporal periphery was an area of grayish-white exudation about 2 disk diameters in extent. The vessels over this area were small, but showed sharp and irregular changes in direction as if affected by local elevation of the retina.

During the succeeding months the exudation in the periphery of the right eye gradually increased; also some definite beading of vessels became evident in that region, and a small amount of hemorrhage appeared within the exudate.

During March, 1942, the right eye was given 930 r of x-ray therapy in divided doses, using 200 KV with a 2 mm. copper and 1 mm. aluminum filter, and with a small aperture to avoid irradiating the lens. In spite of this therapy the area of exudation gradually but steadily increased in size, the disk became much more edematous and a hazy spot of subretinal exudation began to form just below the disk.

The right eye was operated upon on August 1, 1942. The area of exudation in the temporal periphery was completely obliterated and surrounded with 16 diathermy punctures, using Walker pins as in a standard detachment operation. During the first week after operation there was surprisingly little reaction in the fundus. Eleven days after operation corrected vision was still 20/20 -3. There was an elevated white area with some absorbing blood in it extending from 7 to 10 o'clock in the temporal periphery. The disk was slightly more edematous and the subretinal exudate below the disk was slightly more prominent than before operation. Four weeks after operation the fundus reaction reached its height, with more edema of the disk, extending out into the macular region. At this time vision with a pinhole disk was reduced to 20/70. During the succeeding four weeks the area in the periphery that had been treated with diathermy punctures contracted down into a white scar with a pigmented border. The edema of the macula and about the disk disappeared, the subretinal exudation just below the disk absorbed, and corrected vision returned to 20/20 -3.

There has heretofore been no treatment of any kind for juvenile Coats' disease. The ap-

parent success of diathermy punctures in this case is therefore of considerable importance. There has been considerable argument as to whether or not the pathogenesis of juvenile Coats' disease is the same as or similar to that of angiomatosis retinae, but whatever the primary cause of the lesion may be, the observations here reported indicate that obliteration with diathermy at least offers a chance of saving vision. It is significant that in this patient eradication of the primary lesion in the periphery of the retina caused disappearance of the edema of the disk and of the subretinal exudation below the disk.

FREDERICK C. CORDES, M.D., San Francisco, California: It is only recently that the treatment of angiomatosis retinae has received much attention. In addition to the method employed by Doctor Lewis, radium and x-ray have been used. Treacher Collins in 1930 proposed electrolysis as a possible therapy and suggested that localization of the tumor be done according to the methods employed by Gonan. X-ray has been employed a number of times.

There are very few reports of cases followed for more than a year. Weve reported his result two and a half years after diathermy and Craig, Wagener and Kernohan reported their observations three years after x-ray therapy.

In 1940, Doctor Hogan and I reported our results in a fairly early case of angiomatosis retinae in a girl of 20 who received 1202 r of x-ray in September of 1938. In the left eye there was a lobulated, reddish-yellow mass situated well out in the periphery below and slightly temporally. The inferior temporal artery a short distance from the disk was markedly dilated and tortuous. In the central portion the artery became sacculated. Accompanying the artery was a similar but more dilated tortuous vein which started in the mass. There were patches of pale yellow exudate above the macula and a good deal of edema and linear streaks of exudate along the vessels.

The patient was observed periodically until she moved to Seattle. The improvement described in the paper remained and did not retrogress. In Seattle, the patient has been under the observation of Doctor Will Otto Bell, who has been kind enough to keep us posted on the findings when she comes for periodic examinations. His letter three and a half years after irradiation was started states, "The sacculation and tortuosity mentioned in the report have markedly receded. The vein remains somewhat enlarged. The mass measures almost 2 disk diameters in size and roughly shows elevation of from 3 to 4 diopters. No other masses have been seen. The patches of pale yellow exudate above the macula in the original report could not be found and the linear streaks of exudate above the dilated vein are absent." Vision remains the same as before treatment: 0.8 with correction, which was her vision previously. And interestingly enough this reduction in vision is due to a lens opacity that she had preceding irradiation and which has not increased as a result of irradiation.

In addition to the above patient we have under observation a 14 year old girl with bilateral angiomatosis retinae. The condition is well advanced in the right eye and is in the early stage in the left. Her father died at 35 of Lindau's disease. Aside from the eye findings, the patient shows no other signs of the disease.

In February, 1941, she received 1800 r to each eye. (Please note that is twice the dose Doctor Guyton gave his patient.) She was last seen in September, 1942 (2 weeks ago), 19 months after irradiation was started. There has been no improvement in the well advanced stage in the right eye, and the condition seems definitely more advanced. In the left eye the nodule is smaller, white and "harder" as though it consisted for the most part of scar tissue. The vision of the left eye remains at 20/20.

Well advanced cases have been reported in which x-ray therapy has been unsuccessful. In our experience this observation seems to be borne out. In the second case the well-advanced stage in the right eye showed no improvement. The early stages in case 1 and the other eye in case 2 did respond to x-ray therapy and the eyes have maintained their improvement and function, one for three and a half years and the other for one and a half years.

There still persists a rather widespread fear of x-ray therapy in eye conditions. The most dreaded complication of radiation therapy for ocular lesions is lenticular opacities, a condition that can be corrected by operation and therefore should not be considered too seriously. Clapp could find only 34 cases of post-irradiation cataracts up to and including 1932 which covers the period when irradiation of an eye was not as well understood as it is today.

Based on observation of three eyes, irradiated and observed three and a half years in one instance and one and a half year

in the other two, we feel that in addition to diathermy, x-ray does offer a convenient, safe way of treatment of these lesions if they are seen early before gliosis has developed.

F. Bruce Fralick, M.D., Ann Arbor, Michigan: I am pleased that Doctor Lewis has again brought to our attention the fact that angiomatosis retinae in some instances is amenable to treatment. He is to be complimented on his careful observations and results in this case.

It is my belief that the number of patients operated upon for this condition will be small and therefore everyone should place on record their results in each individual operation so that the total of such experiences may be reviewed and evaluated. I wish therefore to add an additional case of angiomatosis retinae operated upon by me.

A 26-year-old male patient was referred to me by Doctor Warren B. Dodge, Jr., of Battle Creek, Michigan. The patient gave the history that he had experienced no visual complaint until eight months previously when he had noted blurred central vision O. D. There were no subsequent visual changes and no inflammation. The general health was good, and the family history was irrelevant. General medical and neurological examination revealed normal findings. The R. V. was 6/30, L. V. 6/4.5—. External and fundus examination of the left eye revealed normal findings. Examination of the right eye revealed normal pressure and no external evidence of disease.

Fundus examination of the right eye revealed a much dilated and tortuous superior temporal artery and vein. The vessels terminated in an angiomatous cyst in the upper outer retinal periphery. The superior temporal quadrant of the retina was studded with small lipoid flecks. The macular area was infiltrated and edematous and contained many lipoid infiltrates. The central field of vision presented a scotoma in the lower mesial quadrant which included the fixation area.

On April 29, 1941, a diathermy operation for angiomatosis retinae was done following the method outlined by Herbert Kaye in the Archives of Ophthalmology 25:443 (March) 1941. An attempt was made to obliterate the angiomatous retinal cyst with micropuncture pins and to sclerose the superior temporal artery supplying the cyst by means of scleral surface diathermy, along the course of the superior temporal retinal artery. This latter procedure we found very difficult since the artery and vein were relatively close together. An attempt was made to produce the fundus picture of arterial rather than venous occlusion.

When the patient was last seen February 17, 1942, or 10 months after the operation, the vision had improved from 6/30 to 6/15 +. The dilated vessels were much smaller than preoperatively, but the superior temporal artery and vein were still patent. The angiomatous cyst appeared as a productive exudative scar supplied and drained by the superior temporal vessels. The lipoid exudates had nearly all disappeared. The retina was flat and the hemorrhages absorbed without evidence of their former presence.

This limited experience with a condition for which formerly no therapeutic measures were available offers hope for further improvement in surgical technic and results.

Arthur J. Bedell, M.D., Albany, N. Y.: My object in coming to the platform is to demonstrate by means of colored fundus photographs that angiomatosis passes through certain stages whether treated or not, and further, that these stages do not fall into definite time intervals.

(Slide.) In the first photograph, there is shown an unusually large vein in the inferior portion of the fundus. I believe it is fairly safe to say that when there are very large parallel blood vessels, angiomatosis is present.

(Slide.) The second photograph of the same eye shows the macular exudate and greater dilatation of both the vein and accompanying artery.

(Slide.) And the third one of the extreme temporal periphery shows a typical angioma; a rounded, pale, red swelling, with a paler area below and a pinker upper outer edge, with entering artery and draining vein.

The second case was in a more advanced stage. The angioma and the blood vessels were small, but the exudate covered a greater area and in the lower part of the fundus there was a detachment of the retina. In the case which Doctor Lewis presented, you remember that following his treatment there was retinitis proliferans. I am sure that when he reports in another year or two, he will tell us that the retinal folding and proliferation have increased.

(Slide.) The photograph of this third case exhibits typical changes with much pigmentation and several retinal ridges.

Angiomatosis is very much more common than the reports indicate. I have seen 20 cases and feel certain that others may have been unrecognized.

INFLUENCE OF WEATHER CHANGES ON OTOLARYNGOLOGICAL DISEASES

WILLIAM F. PETERSEN, M.D.
BY INVITATION
CHICAGO

THERE are fashions in medicine as in other human affairs. These fashions may concern our methods of thinking, our approach to the study of a problem or the technic of an operative manipulation, the use of a particular diet or vitamin or drug. In an era of mass propaganda we, too, are carried away by the bizarre and the novel—the while ignoring the simple and the commonplace.

Lawrence Henderson* in his "Study of Man"[3] expressed this in the following paragraph:

> The disposition to think in this manner (i.e., to think of a phenomenon as simple, as its cause as single, and to feel that, accordingly, there is but one clear, straight path to be followed in action) has also been strengthened both by the conditions of modern life upon the practice of medicine and by increasing specialization. So it comes about that sick people are often regarded as cases rather than persons, and many things in the history of their sickness, many others concerning their environment, which were familiar to general practitioners of an earlier day, are likely to be overlooked and not even suspected. And yet disregard of such factors or of others that seem unimportant in the light of oversimplified theories often leads to disaster. The disregarded factors are, as often as not, among those which the old-fashioned general practitioner intuitively recognized and which can sometimes be discovered only through intimate acquaintance with the patient and familiarity with his environment, with his life day by day, and with his family history.

Presented at the Forty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 11-14, 1942, Chicago, Illinois.

* This paper, delivered at the University of Pennsylvania Bi-centennial, presents a broadly conceived and penetrating examination of the reason for success and failure of modern biological research. It was, unfortunately, his last contribution; Lawrence Henderson died January 10, 1942.

The weather is one such factor in our environment which is today completely ignored by the medical scientists and, of greater importance, by the practitioner—whether family doctor or specialist.[4] That the otolaryngologist should disregard the importance of the changing air mass in which the patient exists is the more interesting because he deals wholly with organs that are directly concerned with the atmosphere.

All animal life depends on air as the prime factor in the environment and on its oxygen as the keystone of the organic chemical processes. But the atmosphere is of significance in almost every other environmental interrelation whether in the transmission of sound and light, of particulate matter (bacteria, pollens, dust, odors), of moisture; its electric charge is of importance; to its pressure and velocity of motion and particularly to its effective temperature the organism must instantly adjust.

The organism adjusts in part and as a whole to changes in the atmosphere. With the immediate reaction of the ear or nose or throat or bronchi the otolaryngologist is obviously concerned and, in this day of the extreme demands of air change entailed in aeronautics, he has become air-minded because of the acute conditions with which he has to deal.

But in the background (although actually of greater biological import) lies the effect of change in the air mass (weather) on the body as a whole and it is this phase that I shall discuss at length.

We live in the region of the circumpolar storm tracks—the "roaring forties"—a mixing zone of cold air (polar air masses, chilled by contact with the poles) and warm air (tropical air masses, warmed and moistened by passage over the vast areas of the tropical seas). These are physically different atmospheres and pass over us as great rotating masses (the warm cyclones, counterclockwise; the cold anticyclones, clockwise). At their interfaces, as we might expect, there is physical disturbance in the form of wind and storms, and precipitation phenomena are common (rain, fog, snow, etc.).

The animal (and plant) world must constantly adjust to these physical changes and the human, because of his naked skin, is particularly sensitive and has developed the integrative reactions (biochemical, endocrine, nervous) to a remarkable degree of perfection. Because of this, man, even in his primitive state, has been able to survive under conditions of extreme cold and extreme heat as well as taxing variability; obviously, such adaptation has been achieved as the result of most vigorous selection and survival.

Modern man, though he has inherited the amazing capacity to adjust has (1) ameliorated some of the strain by perfecting protective mechanisms while (2) on the other hand, his rapid development has exaggerated demands in other ways.

As a result of (1) enervation on the one hand and (2) immensely fatiguing demands on the other, we experience the organic breakdown that we label "disease" (for disease is basically inability to adjust to an environmental situation).

In our region it is the cold air mass that involves greater strain on the human. Against it the organism seeks to insulate itself and does this by withdrawal (peripheral vessels contract, there is less heat loss, catabolism is lessened peripherally, a transient alkalosis obtains, membranes are less permeable). This vasoconstrictive state (adrenal, pituitary, etc.), with increased tension of smooth muscle in general, may roughly be spoken of as a sympathicotonia. It places greater load on the left heart. An anoxia obtains in many organs; the host-parasite relationship in the mucous membranes becomes disturbed.

If this state persists a corrective reaction follows. The period of anoxia with its production of capillary-active substances is followed by a tendency to vascular dilatation, pressures fall (greater load on the right heart), membranes become more permeable, tissues are hydrated, bacterial penetration may be enhanced.

Because of the continuous pendulation of the air masses the organism is subject to a continuous pendulation of its biochemical and biophysical state. As long as the organic reaction keeps pace with the inorganic environment all is well.

Difficulty arises, however, if environmental demand is too great, if the changes are too great, if the organism is either too young or too old to adjust properly, if other rhythms (diurnal, seasonal, menstrual) amplify the response, if other energy impacts (trauma, emotion, work, etc.) act to augment the meteorological effectors.

To illustrate the effectiveness of the change in meteorological environment on the organism, a day-by-day blood pressure record of an old woman kept under constant hospital conditions (constant diet, no emotional disturbances, no undue physical activity) is illustrated in figure 1.

It will be observed that the day-by-day variability is very great; each pressor crest is associated with the passage of a cold air mass over the city.

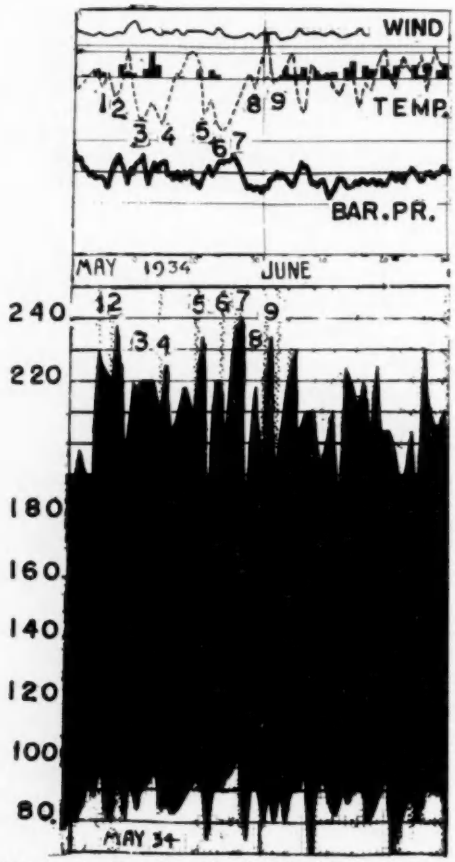

FIGURE 1 — Systolic and Diastolic Blood Pressure Curve. May 1934. Superimposed meteorogram. On this, the wind velocity is indicated on the upper curve; the mean temperature by the dotted line; barometric pressure by the solid line. Passage of polar air masses is indicated by numbers 1 to 10 and correspond to increases in systolic blood pressure similarly numbered.

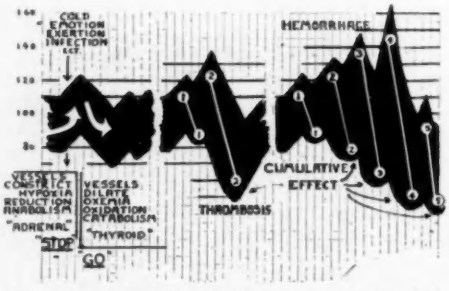

FIGURE 2 — Schematic Blood Pressure Curve. Episodes of increasing pressure (relative anoxia—adrenal phase) followed by periods of decreasing pressure (thyroid phase). Summation episodes (1)-(2) and (1)-(2)-(3)-(4) with pressor crests, followed by fatigue (low blood pressure).

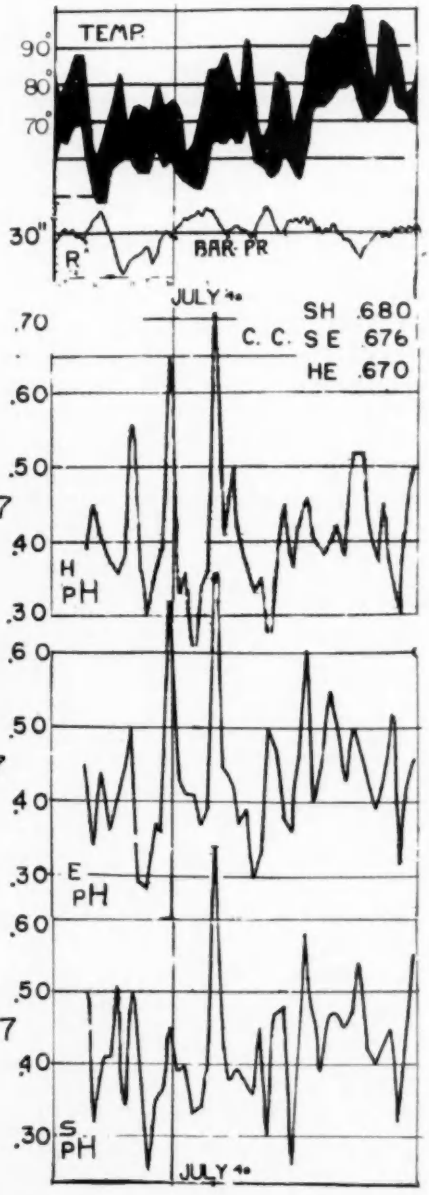

FIGURE 3. Blood pH (Venous Blood of the Arm). Day-by-day observations on adult triplet subjects H., E., and S. June 19 to August 1, 1940. Correlation coefficients are all significant. Meteorogram superimposed; maximal and minimal daily temperature indicated by black solid field, barometric pressure by curve below.

Diagrammatically this pendulation is illustrated in figure 2. Here a single ef-

fector (cold, emotion, trauma) is indicated, with a sympathicotonic response followed by the corrective phase of vascularization.

Summation may occur (as shown in the two following portions of the diagram) when an environmental impact is repeated before equilibrium has been reëstablished or if other effectors impinge. The pressor levels may be pushed to extremes or depressed, but either extreme is fraught with danger.

Reactions such as these involve every organic balance. If, for instance, we follow the day-by-day study of the blood pH level (arm) in a set of adult triplets,[5] the curves in figure 3 are obtained. The correlation coefficients of the interrelations of all the men are significant, i.e., the reactions of these like individuals are practically identical and these are reactions to the air mass in which they were living, each period of lower pH (relative acidity) following a period of relative anoxia due primarily to cold.

CHANGE IN THE NASAL MUCOUS MEMBRANES

In this group of triplets we made daily observations of the nasal pH and in figure 4 the changes in the three men are indicated; again they are alike, with wide range from 5.0 to 8.0.

When the pH of the nasal mucous membranes is subtended below that of the pH of the blood (Subject H) it will be observed that the trends are opposite, i.e., when the venous blood of the arm becomes more alkaline the surface of the nasal mucosa becomes more acid (figure 5).

Here then is evidence that the nasal mucous membranes, like all other tissues of the body, swing in a rhythm that is clearly related to the total organic status, but that in this instance the trends are opposite (i.e., opposite sides

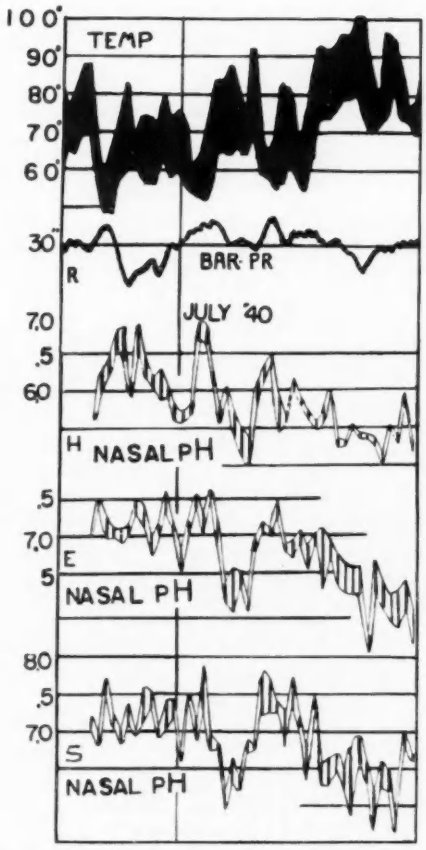

FIGURE 4. Day-by-day Observations of Nasal pH. Triplet group. Meteorogram superimposed. Note corresponding reactions in the group.

of a living membrane) to those of the venous blood of the arm.

In turn this means that the functional state of the mucous membranes not only pendulates but that this in turn is linked with the meteorological environment.

One question that is not solved is the degree to which the reactions of the whole organism are conditioned by the impulses coming from the nasal mucous membranes, which are, after all, the most sensitive organs to come in contact with changing air masses. I suspect that this influence is very great indeed, partly because of the development of the organism and because of the function of

the mucous membranes in connection with the passage of air over the surfaces.

Not only does the function change but the bacterial flora changes.[2] In this same set of triplets Dr. Cahn-Bronner made day-by-day studies of the surface bacteria over a specified area and in the next set of curves (figure 6) these variations in the total number of bacteria that were found can be observed.

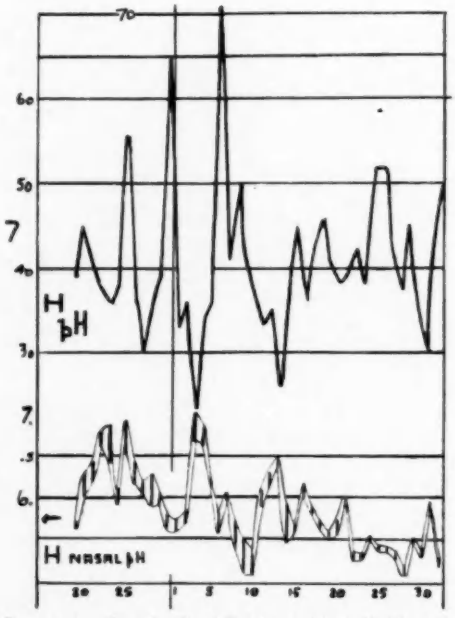

FIGURE 5 — Blood pH of Triplet Subject H (Period July 19 to August 1, 1940). Superimposed over Curve of Nasal pH. Note opposite trends.

Each change in the air mass is associated not only with a change in the functional status (as indicated by the pH) but also by a definite change in the surface bacteria. Again the trends are alike, more bacteria appearing on the surface with periods of greater warmth of the air mass.

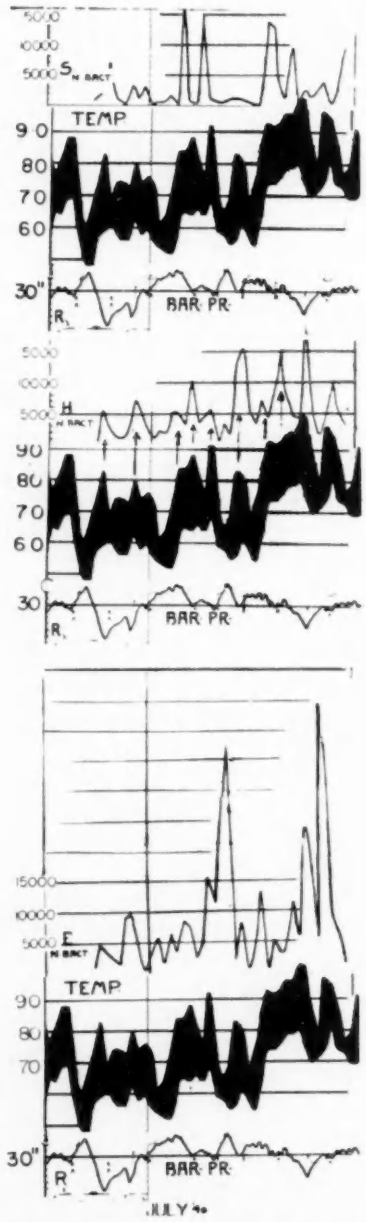

FIGURE 6 — Day-by-day Determinations of Nasal Surface Flora, Triplet Group. Bacterial curve superimposed over meteorogram. Note wide variations in day-by-day count (observations of Dr. Cahn-Bronner).

THE COMMON COLD

In view of the fact that few correlated observations of the nasal mucous membranes and the general environmental situation and the organic status of the patient or the normal are available, it may be useful to present studies

made in a normal white adult male (53 years of age, normosome in habitus, of sedentary habits) who was observed for a four-month period during 1940 and who, during this period, recorded minor illness including the onset of colds and laryngitis.

In the first of the graphs of figure 7 the record of the day-by-day blood pressure (Sundays excluded), and the pH level (venous blood of the arm) are shown. A meteorogram has been extended over the curves.

In examining the meteorogram it will be noticed that the month of January had been quite taxing, with several subzero waves. The month of February reveals considerable variability, barometric pressures ranging from 30.6″ to lows of 29.55″. Temperatures during this time remained fairly stable, although a single decline of considerable extent occurred on the 25th of the month. March revealed the passage of two major polar air masses, with barometric crests on the 11th and on the 25th. Temperature swings were very marked. April, as is commonly the case, revealed wide swings in environmental temperature and marked changes in barometric pressure.

The *blood pressure* (daily determinations of diastolic and systolic blood pressure made at the same time each morning) was unusually stable in this subject and only one period of distinct increase (142 mm. on February 10) and one period of very low blood pressure (a diastolic of 67 mm. on March 13) were observed during this time. Periods of increasing blood pressure occurred regularly with the passage of each cold air mass (ten such episodes are numbered for the month of February) and a corresponding increase in barometric pressure is indicated by corresponding numbers. Inspection makes evident that the reaction of this individual was consistent at all times and corresponded to the change in the air mass.

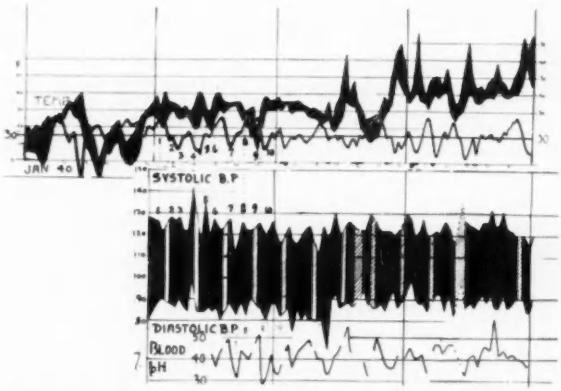

FIGURE 7 — Day-by-Day Observations of: 1. Systolic and Diastolic Blood Pressure; 2. Blood pH (Venous Blood of the Arm). Meteorogram superimposed. Period from January to May 1940. Systolic pressor episodes 1-10 indicated for February, corresponding changes in barometric pressure and temperature of the time as well as in pH levels of the blood.

The *pH levels* of the venous blood always declined after the passage of such polar air masses and for the month of February four such periods of decline are noted and numbered 7-8-9-10, corresponding to the periods of increasing blood pressure which had preceded, and had produced a relative anoxia at the time.

In figure 8 the same two curves, i.e., meteorogram and blood pressure and blood pH are indicated, and below these there has been subtended the *breath holding time* as well as the *CO_2 content* of the blood. Over the top of the curve the periods of *upper respiratory tract disturbances are numbered*. The first "cold," shown by the bracketed number [*1*], occurred in connection with the passage of a cold air mass. The sore throat and cold of early March [*10, 11*] was associated with a period of increasing barometric pressure and a barometric crest. On the 30th of the same month [*14*] another cold was recorded, minor

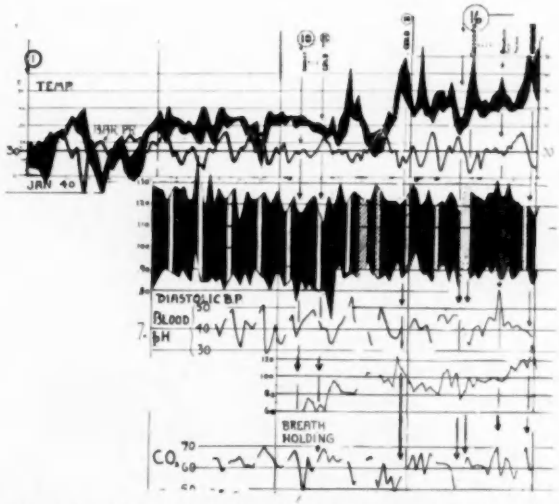

FIGURE 8 — Curves of Previous Group. Periods of upper respiratory tract disturbances numbered (1), (10-11), (14), (16). Corresponding vertical arrows are carried through the graph.

in character; this occurred with unusually high temperatures and a period of relatively low barometric pressure. The much more severe episode, labeled [*16*], which began with throat discomfort on April 13, was associated with the passage of a cold air mass, and with rising temperatures *laryngitis* developed and finally *bronchitis* on the 22nd of April, followed by further symptoms of a cold. A rather severe *headache, with frontal sinus discomfort* was reported on the 29th of April. Down-turned arrows indicate these episodes and it will be noted that the sore throat and cold (episodes 10 and 11) followed periods of sharply declining blood pressures, declining pH levels — in both instances, too, breath holding time was relatively short. With episode [*10*] the CO_2 content had been relatively high in the blood and with episode [*11*] it was increased.

The development of the cold (episode 14) occurred with lowered blood pressures and followed a period of low pH, a decline in breath holding time and occurred with a period when CO_2 content was apparently increasing. The onset of the major disturbance [*16*] was again associated with lower blood pressures, declining pH levels, decline in the breath holding time and with a period when CO_2 had been low but was increasing. The intercurrent change in the clinical condition, the onset of bronchitis, was associated with a crest in blood pH levels and followed a crest in blood pressure; CO_2 content at the same time had been high. Finally the headache on April 29 was associated

with lowered blood pressures, decline in pH levels, a transient decrease in breath holding time and high CO_2 content of the blood.

In figure 9 the curve of the nasal pH* has been extended below that of the maximal and minimal daily environmental temperatures. The first cold [*1*] occurred during a period of rapid rise in pH levels (infrequent observations); episodes [*10*] and [*11*], when the levels were again high; episode [*14*], with a period when the levels were unusually low; episode [*16*] with a low; the onset of the subsequent bronchitis, as well as the onset of the subsequent headache and frontal sinus discomfort were again associated with transitions from high levels to low.

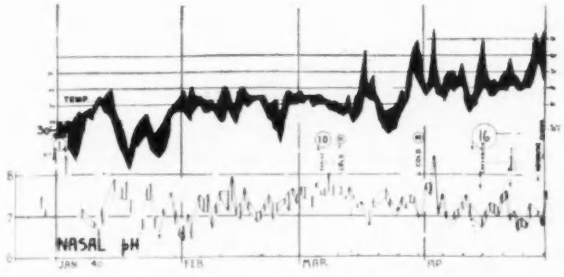

FIGURE 9.—Curve of Nasal pH Determinations Extended over Maximal and Minimal Temperatures and Barometric Pressure Records of the Time. Clinical Episodes Numbered as Before.

This rather complicated record makes evident the close integration of the air mass in which the subject exists with the vascular and chemical state of the blood and mucous membranes, and also with the initiation of disturbances in this balance, i.e., with cold, sore throat, laryngitis, bronchitis, etc.

In connection with the three clinical episodes, identified by the bracketed numbers [*10*], [*11*], [*14*], [*16*], it is of primary importance to note the following:

The episode [*10-11*] occurred with the crest of a period of (A) declining blood pressure levels (fatigue!) which reached their minima on the 5th and 13th of March. These are associated with (B) major disturbances in the CO_2 levels of the blood (March 7).

The (C) breath holding time was shortest on March 10 to 12, and finally, (D) the nasal pH changed from a low on March 4 to a high on March 8.

The second period of disturbance [*14*] occurred at the end of a wide fluctuation of environmental temperature with rise in barometric pressure levels. Blood pressure (A) had declined from the 24th of March; again there was a disturbance of the CO_2 levels with a (B) low on the 29th of the month, a (C) decrease in breath holding time (i.e., a relative anoxia), and finally a (D) change in the pH of the mucous membranes from lows on the 30th of March to a high on April 3.

The third and most significant episode [*16*] was associated first with sore throat, then with laryngitis, finally with bronchitis and followed in the wake of unusually low temperatures, with a polar front which began on the 11th of April and reached its crest on the 12th and 13th.

Again (A) blood pressure levels had declined from a crest on the 9th and 10th to lower levels. The (B) breath

* In determining the nasal pH the glass electrode was inserted in the nose and continuous readings were made for a 20 minute period. Considerable fluctuation may occur during such a time. The degree of this variation is indicated by the vertical line—the curve, therefore, reflects the maximal and minimal level reached each day during such a period of reading.

holding time decreased to a low on the 15th; (C) CO_2 content had been lowest on the 12th and (D) nasal pH had changed from the low of the 13th to a high on the 21st of the month.

Since the CO_2 content of the blood is of great importance in the control of the caliber of the smaller blood vessels, particularly the arterioles and capillaries, and since the CO_2 and pH levels are in turn integrated with the atmospheric status of the time, it becomes evident that the reaction of the mucous membrane (i.e., the physiological state) will be altered. With this alteration the host-parasite relationship is also altered from the norm.

(1) The symptoms that become evident may be on a non-bacterial basis, i.e., purely vasomotor, with changes from relative dehydration to hydration and the consequent interferences with the normal function of the mucous membranes. The patient may have some of the symptoms that are associated with a cold but usually the phenomenon is transient.

(2) One may be dealing with vasomotor plus bacterial changes, when the host-parasite relationship is changed simultaneously, wherein the bacterial phase may appear as the all-important one. Again, as in episode [*16*], a second environmental impact, which will again change the host-parasite relationship, may bring about a change in the symptomatology. In this instance the *bronchitis* actually began when pH levels were unusually high, at a time when environmental temperatures had been suddenly increased, and when there was a marked decline in the pH of the nasal mucous membrane. All these changes, reflecting change in the organic status, are in turn recorded clinically with objective symptomatology on the part of the patient. In other words, the precipitation of the clinical episode, the change in the clinical picture, the appearance of complications and improvement or deterioration are not chance phenomena, but are conditioned by the organic status and this in turn is in a large measure conditioned by the atmospheric environment.

COMMENT

I have discussed the problem of the continuous change in the functional state of the mucous membranes of the nose as they are related to the environment on the one hand and to the general condition of the total organism on the other. I have done this because of its clinical importance.

The "cold" is not only the most common cause of work disability, but it is the precursor of many more serious illnesses—presumably the local change provides the portal of entry for a whole variety of infectious organisms.

The mucous membranes of the upper respiratory tract have as one of their normal functions the destruction of bacteria. For this purpose a group of non-specific and specific mechanisms are available and among them the so-called lysozymes are of particular interest. But these, too, vary in concentration and effectiveness from day to day, and the variation in turn is associated with the pendulation that can be witnessed in the graph.

The common cold is merely one expression of disintegration or disequilibration.[6] The same vasomotor disturbance, irrespective of its origin (i.e., whether due to weather change, emotional upset, fatigue, trauma, etc.) may find its expresson in headache, urticaria, albuminuria, hyperacidity, myalgia, psychopathic episode, spontaneous abortion or biliary colic; it is this that I should like particularly to emphasize, because the specialist, engrossed in his local field of activity, often fails to interpret symptoms that may be referable to other regions. The organism always acts as a whole—nothing happens in one region that is not related to like events in other organs.

Inasmuch as the origin of the common cold is related to the degree of autonomic disturbance and since every such disturbance is diphasic, the investigator may, on careful observation before the onset of symptoms, observe changes that are extreme in one direction or the other: he may observe vasoconstriction or dilatation, a leucopenia or a leucocytosis, a low or a high CO_2 content of the blood, a short clotting time or one that is prolonged, etc.—depending entirely on the particular phase that he happens to observe. Unless aware of this, the whole problem appears most confusing.

Apart from the immediate effectiveness of the environmental impact (whether weather, trauma, emotion, etc.) in swinging the autonomic balance to extremes, the condition of the subject at the time of the impact is of great importance. In the female the sex cycle provides an example, or we may examine the significance of the season.

The periods of transition from winter to summer (March—May) or from summer to winter (September—October) are often critical. This is so because the organism, having once established a modus vivendi in winter or summer, finds any deviation in this established level an unusual strain. The late winter is portentous because the pendulation may swing too far to the acid side (fatigue), the late summer and autumn because the reaction may veer too far to the alkaline side, with accentuation of smooth muscle tone (and undue anoxia). So, too, with warm weather and the skin moist because of perspiration, a seemingly minor breeze may suddenly bring about vast chilling effects on the skin and with marked effects on blood pressure and blood distribution. Though the patient may, at the time, be in good condition biologically, the reaction may be so great that even good resistance may fail critically.

To illustrate this I turn to a case record of Beinfield[1] on "Sinusitis and Staphylococcus Aureus Bacteremia":*

REPORT OF A CASE

F. W., a boy 8 years of age, was first seen by me on August 31, 1940. He had first become sick on *August* 29, complaining of severe frontal headache, general malaise, running nose, redness of both eyes and slight fever. On August 30 his temperature reached 101.5 F., the left eyelids became markedly swollen, the left nostril became obstructed and in general he felt very sick. He had never complained of a postnasal drip or any other trouble with his nose. There was no history of injury. Although he indulged a great deal in swimming and diving during the summer, he had not been in the water for three weeks before this illness. He had not had a cold for eight months. Examination revealed a moderate swelling on the left side of the forehead, extending from the left eyelids, which were tensely chemotic and slightly ecchymotic. The left eyeball was congested but freely movable. There was moderate tenderness in the region over the left frontal sinus and around the swelling on the forehead. The entire nasal mucous membrane was so congested and edematous that it completely obstructed the left naris. Over the left inferior turbinate was a fibrinous exudate, which did not leave a bleeding surface on removal. The right naris was clear. Twelve grains (0.78 Gm.) each of sulfanilamide and sodium bicarbonate were given every four hours (the patient weighed 72 pounds [32.7 Kg.]). Three per cent ephedrine administered in the form of nasal drops and nasal packs failed to produce any shrinkage of the nasal mucous membrane. Inhalations of steam were used with little or no effect. Cold compresses saturated with a 50 per cent solution of magnesium sulfate were applied constantly to the swelling on the forehead.

On *September* 1 all of the symptoms became aggravated, and the temperature rose to 104 F. The patient did not have chills at any time during his entire illness. He was now admitted to the Prospect Heights Hospital. Physical examination gave negative results. The spleen was not enlarged, the neck was not rigid and the Kernig and Babinski signs were absent. Fluctuation was elicited over the swelling on the left side of the forehead; this was aspirated with a heavy needle on a Luer syringe, and 1 cc. of a thick, reddish fluid was obtained and cultured.

* Only one of the series of case records which were presented at the time of the discussion.

Obviously something must have occurred in the environment of this child whereby resistance to an infection became critically lowered. There is nothing in the recorded history that would be helpful. The child had not been exposed, he had not had a previous cold, there was no history of undue exertion.

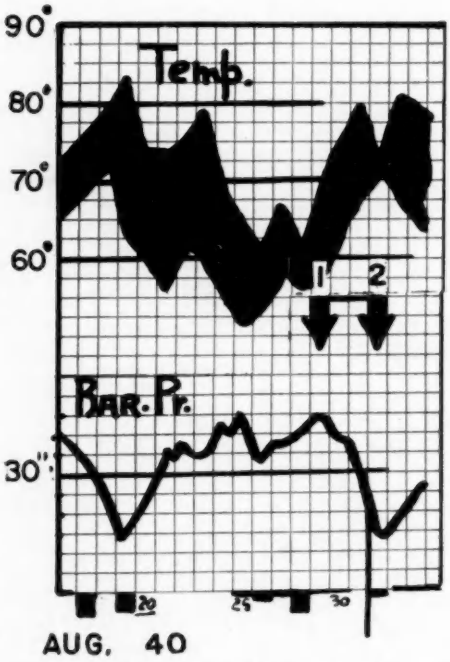

FIGURE 10 — Meteorogram to Illustrate the Onset of the Clinical Episodes [1] and [2] from the Case Report of Dr. Beinfield. Maximal and minimal temperature indicated in the black field, barometric pressure by the heavy black line, precipitation by the vertical columns below the date line. Clinical associations discussed in the text.

So we turn to the meteorological record of the time. In figure 10 the daily maximal and minimal temperatures for New York are indicated in the black field, barometric pressure is carried below in a solid line and precipitation is indicated by the black columns under the date line.

The date of onset is numbered 1 and the accentuation of symptomatology on the 1st of September is labeled 2.

We now note the following:

A severe cold wave had passed; this began on August 19, reached its crest on the 25th. Then there was a secondary effector on the 28th and 29th. During the week of the 25th there was marked precipitation with more than an inch of rain falling on the 28th of the month. Two cold effectors were thus operating, actual fall in temperature as well as lessened sunshine and much rain. On the day of onset there was a barometric crest and on the day of accentuation of symptomatology, a precipitate fall in barometric pressure. The episode [1] would have been associated with vasoconstriction; the episode at [2] with marked fall in blood pressure, swelling of tissues, etc. Another factor: Staphylococci are more prevalent in the summer (and in the tropics); occasionally these are hemolytic.

The constellation of season, of weather, of a characteristic amplification of organic rhythm, with a period of greater susceptibility and finally the coincident presence of the proper bacteria presents a combination that in this instance resulted in infection and accentuation of the infection which had taken place.

While I have made no effort to review the pertinent literature (the material having been presented in lecture form) I should like to call attention to a recent series of papers by Sargent[7] which contribute studies that are of particular importance in connection with the problem of the "cold" and the meteorological environment, season, etc. Having been presented in meteorological journals, they have not received the attention in medical circles that the content would warrant.

This brief outline has been limited to the development of certain general principles and the demonstration of the changes immediately underlying the common cold. The cold is the clinical expression of an autonomic disequili-

bration—primarily a fatigue reaction—in which many factors may play a conditioning role, but cold and environmental variability, at least in our region of the world, are factors that must at all times be considered.

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THE RELATION OF CILIARY INSUFFICIENCY TO DEATH FROM ASTHMA AND OTHER RESPIRATORY DISEASES

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DEATH may result from physiologic failure of any one of a number of systems, such as the urinary, circulatory, or central nervous system. Death may also occur from failure of the ciliary system within the respiratory tract, a fact which has apparently received little, if any, attention. My interest in the subject is rather accidental. Some years ago I published the results of some experimental work on the nose in which one nostril of a group of rabbits was permanently closed surgically.[7] An unexpected change was observed on the closed side. There was a more or less complete metamorphosis of the ciliated epithelium. It had changed to secreting epithelium. The erstwhile ciliated columnar cells were all goblet cells and quite devoid of cilia. This finding was recorded in 1932 without any attempt to explain it. Six years later I sat in on a clinical-pathological conference listening to the pathologist's report on a fatal case of asthma. He described at some length the accumulation of thick mucus in the bronchial tree which had asphyxiated the patient, "because it had been too heavy for the tract to remove." That concept did not agree with the results of some tests that we had done at the Mayo Foundation.[8] We had found thick, tenacious mucus to be the type of secretion which the ciliary system conveyed most readily, especially up vertical surfaces. The pathologist then projected a section of bronchiole upon the screen, and there was the same picture that had occurred in the nasal epithelium of our rabbits behind the closed nostril. Every ciliated cell had become a goblet cell. After the meeting we went over the sections together and found that this metamorphosis was so extensive that there were scarcely any cilia to be found. Apparently the patient became asphyxiated from the secretion, not because it was too heavy to be removed, but because the mechanism designed to remove it had been destroyed.

Since then I have read a number of text books, and only one mentioned the change of ciliated to mucus producing cells.[1] Twelve of the fatal asthma cases reviewed at the University of Minnesota showed this change, but it was mentioned in only one of the necropsy reports.

Huber and Koessler[10] published a very comprehensive article on the pathology of asthma in 1922. They reviewed all the fatal cases described in the literature up to that time (15 in number) and added six of their own, making a total of 21. All these 21 cases had been thoroughly studied clinically and pathologically. Increase of goblet cells in the bronchial epithelium is mentioned in the report of five cases. Two of these were reported by Marchand[14] and three by Huber and Koessler. In each instance it is barely mentioned without description or amplification of any kind. Each of the 21 reports is followed by a comment giving "the most significant pathological changes." In none of them is the change from ciliated to goblet cells listed. Nor in the discussion and summary at the end of the ar-

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ticle is any mention made of this striking change in the epithelium. Fraenkel,[6] in a case report from the year 1900, makes one interesting and significant statement: "The ciliated cells are elongated, and the ciliated border in some is ruptured, and the mucous content protrudes in the lumen like a drop, often fusing with neighboring drops."

Several articles in the literature have made scanty mention of changes in the epithelium since 1922. For instance, Fowler[5] in a caption has this phrase "and hyperactivity of lining cells"; Craige[4] describes degeneration of the mucus glands; Bubert and Warner[3] state that there is "some degeneration and desquamation"; Michael and Rowe[15] state that the epithelium is flattened and thinned; MacDonald[13] speaks of some ulceration and hypertrophy and folding of the mucosa; Lamson and Butt[12] in a caption point out partial desquamation and "rather marked mucoid degeneration" but make no mention of it in the text. Steinberg[16] makes a statement that there is a metaplasia from columnar to squamous epithelium. In two articles there are good photomicrographs of bronchioles that show the metamorphosis, but it is not mentioned by the author in either. Jorgensen,[11] in 1936, reported the pathologic findings in three fatal cases of asthma with meticulous care. He includes a very fine description of this change, and, in my opinion, establishes it as a true metamorphosis and not a replacement of the ciliated cells. Even he, however, fails to mention the loss of cilia and the role that this might have played in the death of his patients.

In a word, this extensive change in the bronchial epithelium that occurs in fatal cases of asthma, although known and described, does not seem to be generally known, nor does its fatal possibilities seem to be in the least appreciated. The loss of ciliary function is not even mentioned in any text book or article which I have been able to find. It seems to me that this subject is worthy of careful study and comprehensive discussion.

This present study has been undertaken in the hope of learning something about the role of the ciliary mechanism in a number of diseases of the lower respiratory tract, such as asthma, acute tracheobronchitis, postoperative atelectasis, influenzal pneumonia, bronchopneumonia, lobar pneumonia and bronchopneumonia, lobar pneumonia and bronchiectasis. Only in the case of asthma are results sufficiently definite so that one can speak with some degree of certainty. Beginnings have been made in some of the others, and some very promising experimental results have been obtained, but only comments in progress can be made on these as yet.

Consider briefly the physiology of removal of secretion from the lower respiratory tract. The ciliary mechanism is similar to that in the nose and sinuses and is doubtless largely responsible, perhaps solely responsible, under normal conditions. Unlike the nose, the lungs are compressible and are in motion. Consequently, a number of other factors may enter in. The lungs when fully expanded contain about 4700 cc. of air. Forced expiration reduces this volume to about 1000 cc. or 1500 cc. Moreover, the bronchioles and bronchi, which cannot be collapsed on account of cartilaginous rings, can be collapsed axially. If there is much secretion in the lungs, this reduction in volume by two-thirds would tend to force it up into the larger air passages. It would tend to cling there during next inspiration, especially if it were mucinous, even though it were forced back somewhat. Coughing is usually very effective in removing excessive secretion. The air is inspired slowly enough so that resistance to its passage is low. It is expired explosively and flows out at such a high rate of speed that the resistance of the

contained secretion is so greatly enhanced that it is carried out by the blast. Air resistance increases as the square of the speed. One gets the impression when making bronchoscopic examinations that the bronchi become constricted during the expiratory phase of a cough. If this is true, then the efficiency of the expiratory blast in carrying away secretion is further enhanced. The principle would be much the same as that involved in the choke of a shot-gun barrel. The push behind the charge of shot is more effective if the barrel is narrowed at the muzzle.

MATERIAL

The material used in this report consists of the records and pathological material from 39 fatal cases of asthma, 12 of influenza, and 10 of bronchopneumonia found in the files of the pathology departments of the University of Minnesota and St. Luke's and St. Mary's hospitals, at Duluth. The experimental animals used were rabbits.

ASTHMA

A review of a number of asthma cases soon convinces one that asthma is not a disease entity but merely a symptom syndrome. This group included quite a variety of diseases. Seven were omitted because they were irrelevant, or because the sections were unsatisfactory. One proved to be a case of acute tracheobronchitis which is discussed separately. The remaining 21 fall into four groups, as far as the pathology of the bronchial epithelium is concerned, as follows: (1) 12 showed the metamorphosis from ciliated cells to mucus-producing, non-ciliated cells, (2) 11 were characterized by surface destruction from bronchitis, (3) five exhibited the marked bronchial constriction that is usually thought to play such a large role in asthma with but little or no destruction of the epithelium, (4) in three, there was pneumonia, with little or no change in the bronchial epithelium.

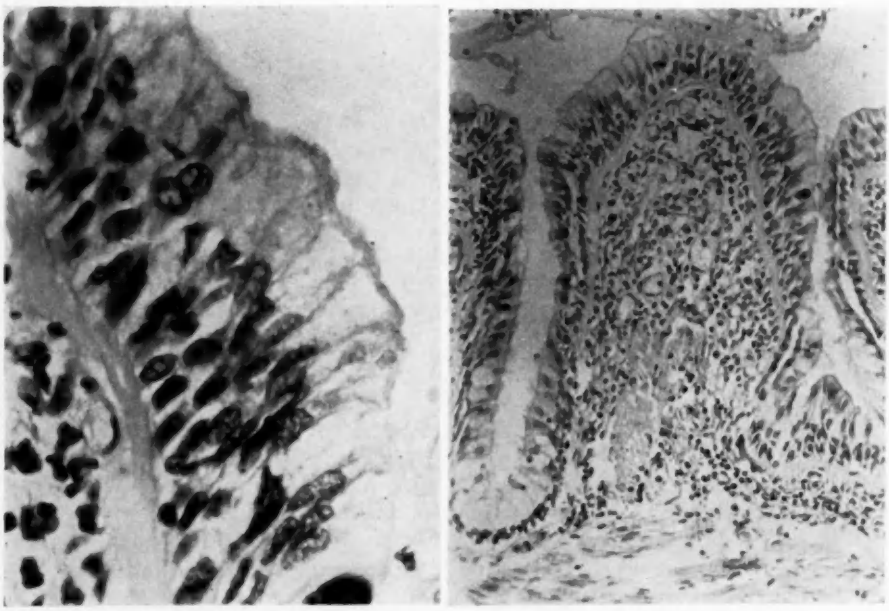

FIG. 1 — Low power and high power views of the bronchiolar epithelium from a fatal case of asthma of six years' duration. Practically all of the cells are nonciliated secreting cells instead of the ciliated cells normally found in this epithelium. A few ciliated cells are found deep in the folds. The folding indicates constriction of the bronchiole.

We are concerned only with the first two groups.

The metamorphosis of the columnar ciliated epithelium seems to begin deeply within the individual cells with the formation of a droplet of mucin (Jorgensen).[11] This droplet enlarges until it eventually lifts the plate of cilia from the end of the cell and escapes. The cilia become lost entirely. (Fig. 1.) Whether the cell then continues to secrete volumes of mucin repeatedly before it is destroyed, I do not know. The secreted mucin does not always escape completely. It protrudes from the cell which produced it, fusing with the mucinous mass in the lumen but still remaining attached to the interior of the cell. (Fig. 2.) Eventually the entire cell seems to melt into or be dragged into the mucinous mass in the lumen. (Fig. 3.) The nucleus may be found at some little distance from the basement membrane. This change does not occur in just a few cells of the bronchial mucosa but involves most of them. In the 11 cases showing extensive metamorphosis the change involved from approximately 30 per cent to 100 per cent of the cells in the areas examined.

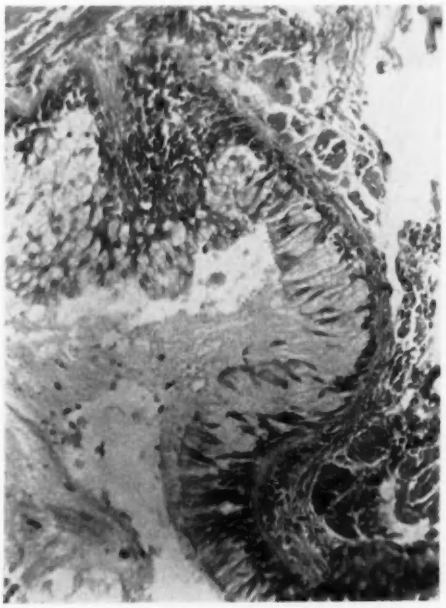

FIG. 2 — The group of epithelial cells in the left center have become mucin producing cells. Note how the mucus strand which has been secreted has fused with the mass in the lumen but remains attached deep within the cells which produced it. Some of the nuclei look as though they had been dragged out of position by traction. There are still some ciliated cells remaining on both sides of the mucus strand.

There were 12 patients in the group which showed the metamorphosis. In 11 it was very extreme, and in the other one, moderate. Death in eight of the 12 was due to respiratory obstruction and exhaustion therefrom. The mode of death is not mentioned in three, and in the remaining one, death was of the pneumonic type. Asthma had been present 11 years on an average—from three to 22 years. Unfortunately the records are incomplete as far as history of allergy and blood counts are concerned. However, nine of the 12 either gave a history of allergy or showed eosinophilia.*

Plugging of the bronchial tree by thick mucinous secretion is described in 10. The air passages were said to be filled "with mucus and frothy material" in one of the remaining two, and, in the other, the pathologist said that the air passages were closed by swollen mucosa. There were no other significant pathological findings in seven of the 12 excepting those of asthma. Active tuberculosis was found in one, and signs of right heart failure in three. Bronchopneumonia was described grossly in two, but the sections did not show any.

It has been suggested that these findings in the epithelium might be due to postmortem change. Postmortem change can be ruled out on the following facts: (1) Necropsy was performed in six of these patients, less than four hours after death—in two, less than 1½ hours after

* All may have shown eosinophiles in the sections, but my attention was on the epithelium, and not having allergy especially in mind at first, I paid scant attention to the eosinophiles. There has been no opportunity to go back to the University to check them again.

death; (2) Islands of well preserved cilia were found in eight of the 12. In postmortem change the cilia are lost uniformly; (3) Postmortem change in the epithelium is fairly typical. The changes found in these cases were of a different type entirely.

The effect of the metamorphosis is twofold. First, an additional amount of viscid mucin is produced. This in itself should not be serious since it is of the type which is removed most rapidly under ordinary circumstances. But, second, the entire ciliary mechanism is lost. What Proetz has called the "escalator" function of the mucosa is gone, and the mucus accumulates until the air passages are full because there are no cilia to remove it. To aggravate matters further for the distressed patient, the mucinous mass remains attached to the interior of myriads of epithelial cells. Actually the mass in the lumen is an integral part of the mucosa. (Figs. 3 and 4.) At necropsy it is often difficult to detach with forceps. Steinberg[16] has pointed out that the mucus plugs are "adherent to the bronchial walls." It seems likely that this firm attachment of the secretion to the mucosa accounts for the ineffectiveness of cough in the asthmatic. The blast of air as it passes over the mucus and through the constricted channels fails to dislodge it, but exerts enough traction to stretch out some of the epithelial cells (Figs. 2 and 3). At the same time the alveolar walls give way under the oft repeated increase of air pressure, and emphysema develops.

Asthmatic patients ordinarily do not asphyxiate in their first attack. They suffer through many of them. One won-

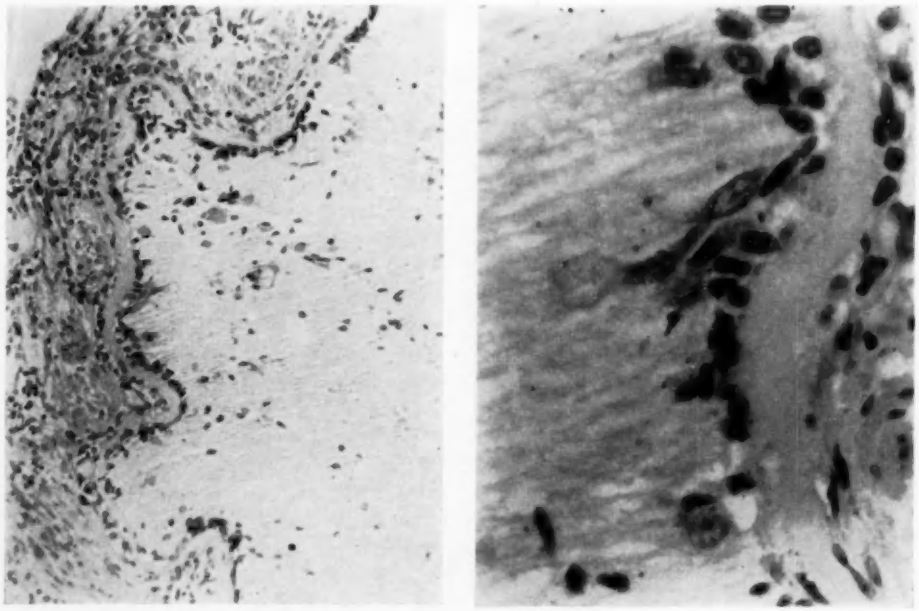

FIG. 3 — Low power and high power views of bronchial epithelium in a fatal case of asthma of 20 years' duration. The entire epithelium is melting into the secretion accumulated in the lumen. There are no ciliated cells whatever in this area. It can readily be understood from this illustration why the mucinous mass contained in the air passages remains in place. There are no cilia to move it. One can also see why it clings to the wall so tenaciously when attempts are made to remove it by aspiration or by means of forceps. It is, in fact, actually an integral part of the mucosa. In this illustration also it looks as though the nuclei had been dragged away by traction from their normal positions in the epithelium.

ders just what happens in the mucosa in these non-fatal attacks. Probably the metamorphosis is not extensive enough to cause death. The goblet cells may be sloughed out as the attack subsides, and ciliated cells may be formed again. We know that in the nose and sinuses the ciliated cells are lost under certain conditions and replaced very quickly. Of course bronchial constriction may be a factor in many or all of these attacks, and the attack grows less acute as the contraction relaxes.

The cause of this metamorphosis is not known. It seems to be related to allergy since allergy, or eosinophilia, is prominent in the history of nine of these 12 patients. This number is too small, however, to mean very much statistically. I have one section of a polyp removed from the maxillary sinus of an allergic patient that shows extensive goblet cell formation. On the other hand, one could scarcely imagine that the metamorphosis obtained behind the closed nostril in the rabbits was allergic in origin. Perhaps stoppage or reduction of air flow is the cause in both the animals and patients, or it is possible the change in the experimental animals has no relation to that found in these patients.

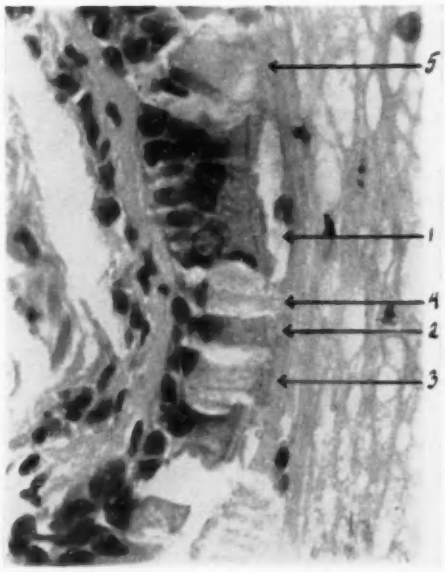

FIG. 4.—From the same case as Fig. 3. Photomicrograph of another area of bronchial epithelium. Here some ciliated cells have been preserved. The mucus in the lumen appears to be free over the cilia but it is attached to the epithelium in the secreting cells. This area suggests that the individual ciliated cells may actually become secreting cells. The different appearances of the individual cells might be different stages in the metamorphosis. Note the differences in the ciliated plate, the protoplasm and the nuclei in the numbered cells.

If the metamorphosis is an expression of allergy, then the asthma which shows this metamorphosis is doubtless a disease entity in which the chief pathological change in this picture is the respiratory mucosa. The other outstanding pathologic features, which are usually considered to be the significant ones, namely, excessive production of mucus, formation of mucus bronchial casts, emphysema, thickening of basement membranes, hypertrophy of muscle, dilatation of bronchioles, could be secondary to the mucosal change. The degeneration of the mucus glands is probably much the same process as that going on in the mucosa.

ASTHMA WITH BRONCHITIS

There were 11 cases in the second group of asthmatics. These were not all similar but they all showed more or less destruction of the ciliated surface cells in the bronchi and bronchioles. The bronchial tree filled up with secretion which, unlike the other group, was characterized by the many pus cells contained. Some of these patients died of asphyxia, as in status asthmaticus, and in these, loss of the ciliary mechanism doubtless played much the same fatal role as in the first group.

The duration of the asthmatic symptoms in this group was 15½ years. There was no clear cut history of allergy in any. Differential blood counts were recorded in seven, only two of which showed any degree of eosinophilia, and in these it was not great—4 per cent in one and 5 per cent in the other. There

is record of a considerable number of eosinophiles in the sections in only one case, and a few in one other. The absence of eosinophiles is noted in some. Seven of these patients died in severe respiratory distress. Two died cardiac deaths, and in two the mode of death is not recorded. At necropsy the pathologist describes plugging of the bronchi in seven. The bronchi contained quantities of pus in three, and in one the mucosa was edematous and congested. Bronchopneumonia was found in three, bronchiectasis in two and active tuberculosis in one.

The bronchial epithelium in this group also was found to be practically devoid of cilia, but the picture was essentially different. There was no extensive change to secreting cells in any of them. (A few goblet cells were noted in two.) There was very extensive loss of the ciliated surface cells by destruction in all. The secretion was characterized by a high content of neutrophiles. In this group, also, death occurred in most instances because the air passages filled up with viscid secretion. This probably could not have occurred had the ciliary mechanism been functioning.

There was one case of acute tracheobronchitis which had been classified as asthma and which deserves some special attention. The patient died of asphyxia, and at necropsy the entire bronchial tree contained thick tenacious plugs of mucus. The cut surface exuded abundant fluid, especially in the upper lobes. This man did not have asthma. He had been ill only three days with an acute illness which began with rhinitis. His first "asthmatic" symptoms appeared just the night before he died, when he developed severe dyspnea. Adrenalin gave transitory relief only. He was hospitalized and placed in an oxygen tent and again given adrenalin without any improvement. Pantopon and atropin

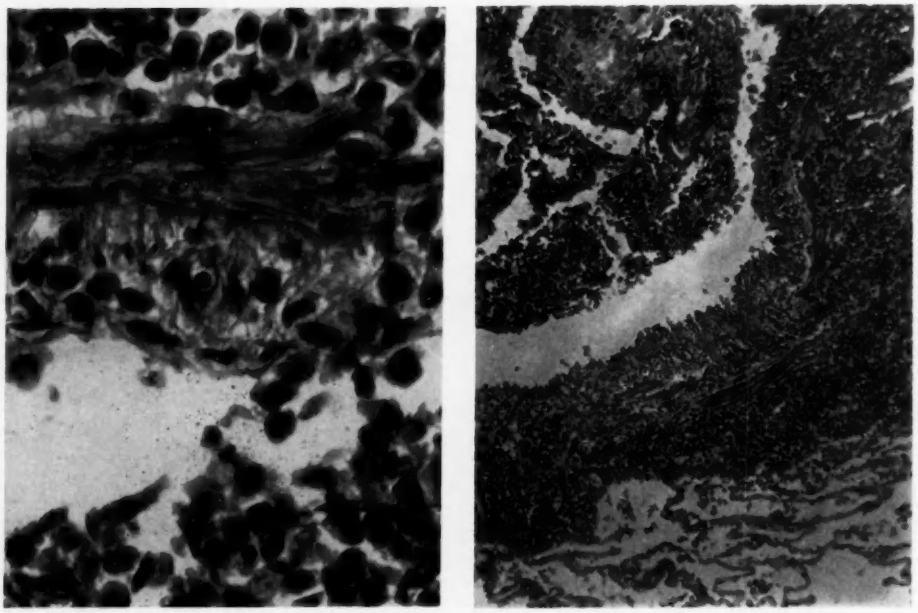

FIG. 5 — Low power and high power photomicrographs of the bronchiolar epithelium from a fatal case of acute tracheobronchitis. There is an extensive severe purulent inflammation with radical changes in the epithelium. The epithelium has lost its columnar form and is exfoliating at the surface. No cilia whatever could be found in this case.

seemed to give some immediate improvement, but he died one hour after this medication. Microscopic examination revealed that the entire bronchial and bronchiolar epithelium in the sections available had been sloughed off down to the last layer of stellate cells next to the basement membrane. (Fig. 5.) There wasn't a vestige of a cilium anywhere. The lumina were filled with purulent exudate. This patient would probably not have died of asphyxia had the ciliary mechanism been there to remove the thick secretion. Conceivably, aspirations through a tracheotomy might have saved him.

INFLUENZA

Twelve cases of influenza from the files of the Department of Pathology at the University were examined. All these patients perished in the pandemic of 1918. The histories indicated that the patients died deaths which were currently described as "drowning in their own secretions." These patients generally filled up with a thin frothy secretion and died after brief illnesses. At necropsy the lungs were edematous and exuded copious quantities of watery, blood-stained fluid on section. There were only a few lung sections saved from each case, and most of these were taken near the periphery. Some bronchioles were found in each, however. The essential picture in these sections was that of acute bronchitis with beginning pneumonia. The bronchioles were filled with exudate, and the epithelium was very badly damaged. (Fig. 6.) In two cases the epithelium was destroyed to the last cell as far as could be determined. It was largely gone in seven, and in two more, partially gone. In only one case was the epithelium fairly intact, and in this one the surface cells were so generally exfoliated that only a few cilia could be found.

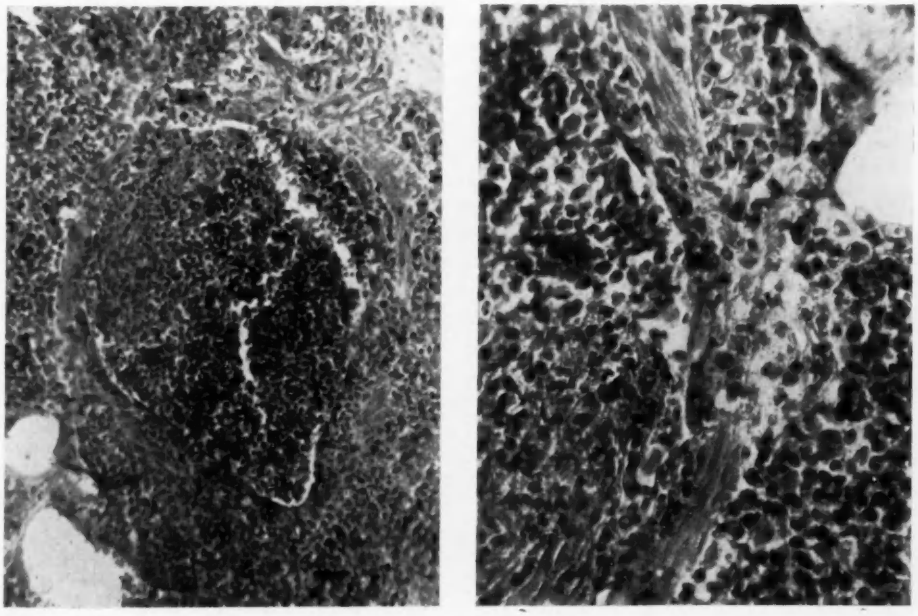

FIG. 6 — Photomicrographs of a bronchiole from a fatal case of influenza from the pandemic of 1918. The epithelium is entirely destroyed, and the lumen is filled with a purulent secretion. This type of secretion can be moved up vertical surfaces by ciliary action when the ciliary mechanism is intact, but not as rapidly as the viscid, mucinous type. In this case all the cilia were destroyed. This was doubtless a factor in the accumulation of the quantities of secretion in the air passages. How important this factor might be is unknown.

We[9] have learned that during the common cold there is very extensive loss of ciliated cells in the nose during the acute stage. In fact it is so extensive that the surface seems to be all but completely denuded of cilia. Living ciliated cells are found floating free in the secretion in untold thousands during this stage. In a day or two, as soon as the acute stage is over, the ciliated cells are found to have been restored.

If this temporary loss of ciliated cells obtains throughout the bronchial tract in influenza (or any other condition) as it does in the nose during rhinitis, then it is quite conceivable that the resultant loss of ciliary action might be a potent factor in the accumulation of such quantities of fluid secretion as to drown the patient. The copious secretion from acute rhinitis pours from the nose by gravity. The secretion produced during acute influenza bronchitis accumulates by gravity in the lower part of the tract, unless it is lifted out by ciliary action, or is coughed out, or removed artificially. However, this very fluid secretion is the type which is moved most slowly up vertical surfaces by ciliary action. All but a thin capillary sheet slides off. Perhaps the production of secretion was so great in those influenza patients that they would have drowned even with an intact ciliary mechanism. Almost surely, however, the loss of ciliary action was a factor. How great a factor it was must remain in the field of conjecture, awaiting more study. It would seem that these cases would have been ideal for aspiration. The secretion was so liquid that it could easily have been removed. If the ciliated cells were replaced quickly, as in the nasal mucus during the common cold, it should have been comparatively easy to tide these patients over the dangerous period.

BRONCHOPNEUMONIA

The question arises, does loss of the ciliary mechanism play a role in the development of pneumonia? It is not usually supposed that it does.[2]

The records of 10 patients dying of bronchopneumonia were reviewed, and the microscopic sections of the lungs examined. In these sections, there were all degrees of pneumonia and all degrees of destruction of bronchial epithelium. In most of the cases, there were areas where the bronchial epithelium looked normal and other areas where it was completely destroyed. The order of events could not be determined. I do not believe that the question can be answered from the ordinary necropsy material. A single case studied very thoroughly, taking serial blocks through the entire tract for microscopic examination, would be more apt to give the desired information.

EXPERIMENTAL WORK

If loss of ciliary action be a factor in the development of lung pathology, then destruction of ciliated epithelium in the experimental animal should produce changes below the destroyed area. I showed some instances of experimental sinusitis two years ago which had been produced by destroying the epithelium at the osteum. If the cilia are destroyed over an annular area in a bronchus, what will happen distal to that point? A number of experiments have been done on rabbits, attempting to destroy the bronchial epithelium through a bronchoscope. Several methods have been tried, most of which did not yield results. It is not easy to destroy the epithelium completely without also destroying other structures as well. Fatal pneumonia, bronchiectasis and emphysema have been produced in a few rabbits, however, by endoscopic manipulation, and results look promising. Beyond that one could not say anything at this time. It is by no means certain that the results were due primarily to the loss of cilia.

TABLE 1

Brief abstracts of history and findings in twelve cases of asthma which were cha-acterized by metamorphosis of the bronchial epithelium from ciliated columnar cells to goblet, or mucin producing cells. Average age was 51 years. The average duration of the asthma was 11 years. Nine contain indications of allergy (history or eosinophiles in blood or sections). Eight of the patients died of respiratory obstruction and exhaustion. One died with pneumonia, and the mode of death was unknown in three. The necropsy reports contain description of plugging of bronchi with thick, viscid mucus in 10 of the 12. In one the air passages were "filled with mucus and frothy material," and in the other, the air passages were "closed by swollen mucosa."

The metamorphic change was very marked in 11, and moderate in the remaining one.

	CLINICAL NOTES						NECROPSY NOTES			
Number and Initials	Age and Sex	Time Elapsing Between Death and Necropsy	Years of Asthma	Course of Disease	Allergy	Eosinophilia	Mode of Death	Lungs Gross	Lungs Microscopic	Other Significant Findings	Metamorphosis
1. E. A. K.	47 F	1½ hours.	3	Suffered from food allergy and was in a state of malnutrition when attack began one week ago. Day before death went into state of status asthmaticus from which she did not recover.	Yes		Respiratory obstruction.	Mucus plugs filling all small and medium sized bronchi. Difficult to remove by pulling with forceps. Mucosa pale. Emphysema marked.	Bronchial epithelium shows marked metamorphosis to to goblet cells ranging up to 100% in most areas. In a few areas, ciliated cells remain and are well preserved Other areas, the entire epithelium is melting into secretion. The lumena are packed tightly with mucus which is continuous with the interior of the epithelial cells.		Marked.
2. M. B.	27 F	7 hours.	22	Hospitalized four years ago on account of asthma. Known to have been sensitive to silk, plants, animal emanations, raspberries, strawberries, lamb, and pineapple. No history of final illness.	Yes		Unknown.	Mucus plugs filling the medium and small bronchi. Viscid gray or white material. Marked emphysema.	Very marked metamorphosis of bronchial ciliated epithelium to goblet cells. In one fairly large bronchiole practically the entire mucosa is melting into the secretion. Mucinous secretion packs the lumen and is continuous with the interior of the epithelial cells. A few ciliated cells are found intact. State of preservation good. Many eosinophiles in submucosa.	Active t.b. in apex of right lung.	Marked.
3. W. D. B.	47 M	15 hours.	5	Very severe attacks of asthma during last 3 weeks. Three days ago felt so much better that he took a walk of 6-8 blocks, following which he was seized with a dyspneic attack which lasted 12-14 hours.	?		Respiratory obstruction.	Muco-purulent material plugging some of the bronchioles. Both lungs light and feathery. Body embalmed.	Very intensive metamorphosis of bronchial ciliated epithelium to goblet cells. In some areas it involves 100% of the cells. Some ciliated cells remain intact in other areas. Where found, they are well preserved. Marked infiltration of eosinophiles around bronchioles.		Marked.

	CLINICAL NOTES						NECROPSY NOTES				
Number and Initials	Age and Sex	Time Elapsing Between Death and Necropsy	Years of Asthma	Course of Disease	Allergy	Eosinophilia	Mode of Death	Lungs Gross	Lungs Microscopic	Other Significant Findings	Metamorphosis
4. J. S. B.	68 F	14½ hours.	6	Asthma at all seasons. Lately more or less constant. 2 or 3 attacks daily and often at night. Characterized by intense dyspnea and cyanosis. Day of her death thought that she was getting a cold. Was put to bed at 6 p.m. and given codein. Attack of severe dyspnea and extreme cyanosis at 9 p.m. Became unconscious and died.			Respiratory obstruction.	Smaller bronchi almost occluded by thickening of mucosa. Lungs light and feathery. Body embalmed.	Very extensive metamorphosis of bronchial epithelium to goblet cells, 30 to 100%. The epithelium lies in folds. Many eosinophiles in the submucosa. Ciliated cells present in some areas and are fairly well preserved.		Marked.
5. N. C.	47 F	2½ hours.	14	Asthma 14 years. Maxillary sinuses operated five years ago without relief. Tonsillectomy same year. One year ago had ethmoid exenteration. Had been using adrenalin every 3 hours. Admitted this time for bronchoscopic examination. At end of endoscopy, 20cc lipiodal injected. Death in five minutes.	Yes	No.	Respiratory obstruction. Heart beat for some time after respiration had stopped.	Solid mucinous plugs closing nearly all bronchi. Mucosa injected and covered with sticky secretion. Emphysema throughout.	Very marked goblet cell change in bronchial mucosa. Lumena filled with mucin which is continuous with the interior of the epithelial cells, these cells being pulled loose from the epithelium by traction. Some cilia found. These are well preserved. Many eosinophiles.		Marked.
6. M. B.	54 F	1 day or less.	20	Asthma 20 years in attacks. Family history of asthma. Worse since she had "flu" six years ago. Had chronic tonsillitis and sinusitis with polypi. Operated with relief for one month. Then recurrence with several attacks, so severe that she was thought to be dying. Adrenalin "fast" last two days.	Not mentioned.	33%	Respiratory obstruction.	All bronchioles filled with thick tenacious plugs. Lungs weigh 300 gms. each. Moderate emphysema. Body embalmed.	Goblet cell change in bronchial epithelium varies from a few up to 100%. Cilia present in some areas and are well preserved. Lumena filled with mucus which is continuous with the interior of the epithelial cells. In some areas the entire epithelium is melting or being dragged into the secretion.		Marked.
7. W. H. S.	50 M	5½ hours.	?	Admitted to Veteran's Hospital on account of asthmatic attacks. Worse in cold weather. Died in attack of dyspnea after six weeks in hospital.	Not mentioned.		Respiratory obstruction.	Entire bronchial tree from trachea to bronchioles plugged with thick tenacious mucoid material. Weight of lungs 335 and 320 gms. Some emphysema.	Extensive goblet cell change. As much as 100% in many areas. Lumena filled with mucin which is continuous with the interior of the epithelial cells. Epithelial cells disintegrating or being dragged into the secretions. Few cilia found. These were fairly well preserved.		Marked.

	CLINICAL NOTES						NECROPSY NOTES			
Number and Initials	Age and Sex	Time Elapsing Between Death and Necropsy	Years of Asthma	Course of Disease	Allergy	Eosinophilia	Mode of Death	Lungs Gross	Lungs Microscopic	Other Significant Findings	Metamorphosis
8. A. R.	43 F	10½ hours.	4	Asthma in attacks for four years. Hay fever every summer for many years. Sensitive to many fruits and plants. Bilateral maxillary sinusitis. Skin lesions during last month.	Yes	17 to 28%	Respiratory obstruction.	Larger bronchi contains thick mucus. Medium and small bronchi completely plugged with it. Few areas of bronchopneumonia in right lung. Wts. of lungs, 830 and 495 gms.	Extensive goblet cell change of bronchial epithelium. Lumena filled with mucus which is continuous with the interior of the cells. Marked loss of epithelial cells into the secretion. No ciliated cells found. Many eosinophiles.	Moderate hypertrophy of right heart. Moderate bronchopneumonia.	Marked.
9. C. A.	60 F	3½ hours.	?	Suffered from recurrent asthmatic attacks for years. Present attack began 2 weeks ago. Adrenalin had little effect. Died in oxygen tent in status asthmaticus.		No	Unknown.	Large and medium bronchi-filled with thick mucus. Small ones below 1 m.m. in diameter do not show this. Marginal emphysema. Ghon tubercle.	Extensive goblet cell change of bronchial epithelium. Lumena filled with mucus which is continuous with the interior of the epithelial cells. Epithelial cells melting into the mucus in the lumena.		Marked.
10. A. R.	64 M	1 1/6 hours.	17	Asthma for 17 years. Urinary difficulty past two weeks. Comatose last two days.		No	Unknown.	Practically all bronchi filled with thick, white mucus. Several small areas of consolidation in both. Wt. of lungs, 470 and 380 gms.	Marked goblet cell change of bronchial epithelium. Varies from a few up to 100% in different areas. Some ciliated cells remaining. Cilia well preserved. Pus and mucus in bronchial lumena. No eosinophiles.	Congestive heart failure. Organizing pneumonia. Chronic bronchitis?	Marked.
11. C. N.	35 M	3½ hours.	3	Asthma for three years. Worse in hot weather. Relief from adrenalin. Increasing asthma last week of life. Broncho-pneumonia with fever and leucocytosis beginning two days before death.		4%	Respiratory failure like pneumonia.	Secondary and tertiary branches of bronchi filled with stringy gelatinous mucus. Lobar pneumonia in left lower.	Material unsatisfactory. One area of epithelium found which looks like bronchiolar. Is 100% goblet cells.	Hypertrophy and dilation of right heart. Acute passive congestion of spleen, liver and kidneys.	Marked.
12. D. C. S.	66 M	4 hours.		Entered Veterans' Hospital on account of chronic bronchitis, asthma, and general arthritis. Had dyspnea, wheezing cough. X-ray showed emphysema. Had attack like asthma.		10%	Not mentioned.	Bonchi and bronchioles filled with mucus and frothy material. Marked emphysema.	Extensive folding of bronchial epithelium with grade II goblet cell formation.	Chronic bronchitis Bronchopneumonia Chronic hepatitis II.	Moderate Grade II.

TABLE 2

Brief abstracts of history and findings in 11 cases of asthma which were characterized by destruction of the ciliated cells in the bronchial mucosa and signs of bronchitis. The average age was 57, and the average duration of the asthma 15½ years. There was no definite history of allergy in any. Two showed low grade eosinophilia (4 and 5 per cent). Seven died with respiratory obstruction, and two with cardiac failure. Mode of death not recorded in the other two. The bronchial tree was found plugged or filled with thick secretion at necropsy in seven. In three there was pus in the bronchi, and in one the "mucosa was edematous and congested." There was no marked metamorphosis of the bronchial epithelium in any although two contained a few goblet cells.

	CLINICAL NOTES						NECROPSY NOTES			
Number and Initials	Age and Sex	Time Elapsing Between Death and Necropsy	Years of Asthma	Course of Disease	Allergy	Eosinophilia	Mode of Death	Lungs Gross	Lungs Microscopic	Other Significant Findings	Metamorphosis
13. J. P.	68 F	4¾ hours.	35	Many severe attacks of asthma. Was in hospital 1 week 4 months ago for status asthmaticus. Was adrenalin "fast." Improved then with combination of diathermy, aminophyllin and adrenalin. Had bronchiectasis and hypertension. Blood pressure 170/104.	No hay fever.	10%	Heart stopped first.	Much cloudy mucus in medium bronchi in all five lobes. Emphysema grade II.	Broncho-pneumonia in many areas. Purulent exudate in bronchi. Marked folding of basement membrane. Considerable loss of surface cells in bronchial epithelium. Cilia well preserved in some areas. Only one small area of metamorphosis.	Broncho-pneumonia.	Few goblet cells.
14. A. S.	57 F	16¾ hours.	20	Has had asthma for 20 years. Almost continuous attacks for past 6 weeks. Very severe attack with cyanosis 4 days ago. Drugs give little relief. Very little air entering lower lungs. Blood pressure 170/95. Had CO_2 and helium. Adrenalin "fast."	Skin tests negative.	4% and 0%	Respiratory obstruction.	Bronchioles 1-2 m.m. all contain plugs of thick, tenacious mucus.	Very extensive loss of surface cells in bronchial epithelium. Bronchioles filled with mucus in layers. Contains many epithelial cells, some p.m.n.s., few eosinophiles. Desquamation almost complete in larger bronchioles and bronchi. Only one small area of cilia found.	Sinuses contain mucus and polypi. Epithelium is atrophic and without cilia.	None.
15. N. S.	57 F	9 hours.	8	Has had asthma for eight years. Final fatal attack began 6 days ago. Brought to hospital two days ago in shock. Blood pressure low. Heart block. Had been taking adrenalin every two hours.	No mention.	None 0%	Cardiac failure.	Bronchioles all contain mucus plugs. Marked emphysema.	Few goblet cells. Cilia well preserved in some areas. Loss of surface cells by desquamation marked in other areas. Laminated mucus with many eosinophiles in bronchus. Other bronchi empty.	Heart chamber dilated. Scar in intraventricular septum.	Few goblet cells found.

	CLINICAL NOTES					NECROPSY NOTES				
Number and Initials	Age and Sex	Time Elapsing Between Death and Necropsy	Years of Asthma	Course of Disease	Allergy	Eosinophilia	Mode of Death	Lungs Gross	Lungs Microscopic	Other Significant Findings	Metamorphosis
16. M. P.	76 M	16 hours. Embalmed.	16	Asthma for 16 years. Worse during past 10 years. Relief from adrenalin until last 48 hours when he was adrenalin "fast." Severe dyspnea afternoon of death. Was sitting on edge of bed when he collapsed.	No mention.	No count.	Respiratory obstruction.	Bronchi and bronchioles all filled with tenacious mucus. Trachea, free. Emphysema.	Bronchial epithelium shows degeneration and sloughing at surface. No cilia found. No goblet cells. Lymphocytes, eosinophiles, and p. m. n. s. in lumena.	Ca. of prostate.	None.
17. H. H.	56 M	3½ hours.	6	Broncho-pneumonia 6 years ago following which he had cough and wheezing. Severe dyspnea and weakness during past 3 weeks. Much viscid greenish yellow sputum. X-ray evidence of bronchiectasis. Sinusitis.	No mention.	10%	Respiratory obstruction.	All bronchi and bronchioles full of mucus plugs. Very tenacious. Lungs voluminous. No consolidation.	Bronchial plugs found to consist largely of inflammatory cells (p.m.n.s.). The epithelium shows extensive loss of surface cells.		None.
18. W. N.	58 M	3 hours.	3	Asthma began 3 years with bronchitis and broncho-pneumonia. Had 50 canaries, 4 dogs, and several cats. More attacks at home than at work.	Possibly?	No count.	Respiratory obstruction and fever.	Bronchi hemorrhagic. Broncho-pneumonia. Pus in bronchioles.	Bronchial walls congested and show hemorrhages. Some broncho-pneumonia. Considerable sloughing of surface cells from the bronchial epithelium.	Acute bronchitis. Congestive heart failure. Hypertrophy of right heart.	None.
19. E. W.	48 F	12 hours.	30	Productive cough and dyspnea for thirty years. Studied for allergy and t. b. with negative results. Dyspnea and edema of legs for past month. Marked cyanosis past 12 hours.	None.	2%	Respiratory obstruction and cardiac decompensation.	Bronchiectasis present. No emphysema. Some broncho-pneumonia. Much pus in bronchi.	Extensive surface sloughing of bronchial epithelium. Lumena filled with p.m.n.s.	Cardiac decompensation. Bronchiectasis. Hypertrophy rt. ventricle.	None.
20. W. C.	56 M	7¾ hours.	7	Dyspnea for seven years. Much worse during past eight months. Weakness and productive cough. Physical signs of asthma.	No mention.	1%	No mention.	Larger bronchi filled with viscid purulent secretion. Purulent exudate can be squeezed from smaller bronchi.	Bronchial epithelium shows hemorrhages, pus, and sloughing of epithelium.	Broncho-pneumonia. Purulent bronchitis. Mitral valve defect.	None.

	CLINICAL NOTES						NECROPSY NOTES				
Number and Initials	Age and Sex	Time Elapsing Between Death and Necropsy	Years of Asthma	Course of Disease	Allergy	Eosinophilia	Mode of Death	Lungs Gross	Lungs Microscopic	Other Significant Findings	Metamorphosis
21. A. S.	33 M	15½ hours.	5	Asthma began after pneumonia five years ago. Last three years has had it almost constantly. Present attack began nine days ago. Came to hospital acutely ill with dyspnea and cyanosis. Disoriented. No fever. Poor response to adrenalin. Given a dose intravenously and breathing became more difficult. Died in less than an hour.	No mention.	No count.	Respiratory obstruction.	Bronchi filled with thick mucus plugs. Consolidation in left base. Emphysema. Lungs do not collapse.	Very extensive and severe sloughing of the bronchial epithelium.	Bronchopneumonia. Acute dilatation of right heart. Suppurative appendicitis.	None.
22. E. T.	54 F	3¼ hours.	26	Asthma and pleurisy for 26 years. Had had t. b. Somewhat better for 5 years after eliminating sweets and pastries. Worse again past 3 months. Dyspnea lately very severe. Especially expiration was difficult. Three days ago entered status asthmaticus. Did not improve again.	?	No count.	Respiratory obstruction.	Bronchi dilated. Contain pus. T. B. with cavitation in left apex. Pleura roughened by old adhesions. Lungs emphysematous and voluminous.	Extensive sloughing of bronchial epithelium.	Bronchiectasis Pulmonary t. b. with cavity.	None.
23. P. O.	68 M	15 hours.	15	Asthma for 15 years. More severe in summer. During past 3 months medications have not relieved him much. In hospital was relieved by ephedrine powder spray for a time. Finally died in extreme dyspnea.		5%		Mucosa of bronchi edematous and congested. Marked emphysema.	Chronic bronchitis (pathologist). Very extensive sloughing of bronchiolar epithelium—grade IV.		None found.

SUMMARY AND DISCUSSION

There is a group of cases among patients who die from asthma which shows a very striking and characteristic change in the bronchial epithelium. This change has been noted briefly in the literature a number of times and has been rather fully described, but it does not seem to be understood nor its importance appreciated. It is entirely ignored in the great majority of descriptions of the pathology of asthma. The change consists in the substitution of goblet or goblet-like cells for the normal columnar ciliated cells. Apparently it is a true metamorphosis. As the metamorphosis takes place, the ciliary mechanism is lost, and the characteristic viscid, mucinous secretion accumulates in the air passages. Normally, this viscid type of secretion is very readily carried up vertical surfaces by ciliary action. The amount of secretion is enhanced, because the erstwhile ciliated cells are secreting as well as the glands. The difficulty of removal of the secretion is aggravated, because the mucin remains attached over large areas within the cells which produce it, thus anchoring the mass to the wall. When the air passages have become sufficiently filled, the patient dies of asphyxia. In my opinion, this metamorphosis is the chief pathologic change and in my opinion death results directly from the loss of ciliary function.

There is a second group of asthmatics in which the cilia are also lost, but the picture is essentially different. In this group, chronic bronchitis with purulent secretion is prominent. There is destruction at the surface of the bronchial epithelium, and the ciliated cells have sloughed off very extensively. In these patients, also, the air passages filled up with secretion to such an extent that the patients died. Presumably the stasis and accumulation occurs largely because of loss of ciliary action.

Bronchospasm must not be forgotten. It was doubtless a factor in some of these cases. In one of the groups which was omitted from this discussion, spasm seemed to play a dominant role.

In the single case of tracheobronchitis which was found the patient also died in extreme respiratory distress. In this patient, the ciliated epithelium was practically entirely destroyed, and, according to the necropsy notes, all the bronchi contained viscid plugs of secretion. Presumably, this secretion could have been removed by ciliary action had the mechanism not been destroyed. In the pandemic of influenza in 1918, many patients died in acute respiratory distress with the air passages and lungs filled with a thin, frothy, liquid secretion. The pathologic material from the lungs of 12 of these patients was reviewed. The material was not entirely satisfactory, but, as far as could be determined, the bronchiolar epithelium had been entirely destroyed, carrying all cilia with it. This type of secretion is not removed as rapidly by ciliary action . . . at least against gravity . . . as are the more viscid types, but undoubtedly the loss of ciliary action was at least a factor in the accumulation of quantities of secretion in the air passages. How important this factor was, must remain a matter of conjecture awaiting further study.

In all these groups, the two types of asthma, the tracheobronchitis and the influenza, I believe that mechanical removal of secretion as a substitute for ciliary action is indicated. Aspiration, either through a bronchoscope or tracheotomy, would be the method. It has been used more or less successfully in asthma for years and more recently has saved the lives of patients ill with acute laryngotracheobronchitis and postoperative atelectasis.

A beginning has been made to study the role of loss of ciliary function in the

pneumonias. Nothing positive can be reported at this time. In a group of bronchopneumonia cases which were reviewed, the necropsy material was found to be unsuitable. The only way in which suitable material may be obtained would be to section the lungs specifically for the purposes of this study.

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DISCUSSION

A. W. Proetz, M.D., St. Louis, Missouri: I think that Doctor Hilding has rung the bell again. When Doctor Hilding talks I always listen carefully because somehow his observations seem to stand the test of time. This is another significant one.

I agree with him that it is, indeed, strange that no one has seen this thing before. Here is just another example of one having to be conditioned to see what is before him. We have never been taught to respect the cilia. We were told that they are a fragile, evanescent sort of thing that may have a function in the lower animals but are negligible in man. Doctor Hilding has been looking at cilia for years and has been thinking in terms of cilia, which accounts for his intelligent reaction to this particular autopsy.

I have been very much intrigued by this theory of his, because I have been rather timidly suggesting for some time that there is a very definite parallel between bronchiectasis and chronic sinusitis and that the two conditions exist for much the same reason. Sinuses are somewhat more rigid than the bronchial tree. Both have the various characteristics which Doctor Hilding mentions. The epithelium at the bronchi is supported by a cartilaginous underpinning which makes it resemble the bony background in the sinuses. As soon as there is persistent obstruction in either case there is trouble. If a little obstruction exists it is very difficult to prevent it from becoming greater, and soon cause and effect react upon one another in a vicious circle.

I wonder whether the change in the cells is actually a metamorphosis or whether the process is a degenerative one. It is important that we should know this. If it is a degenerative process, we have to prevent it. If it is a reparative process, we have to assist it.

I believe it is a degenerative process and results in the formation of this sticky mucus.

I congratulate Doctor Hilding on his very fine paper.

THEODORE WALSH, M.D., St. Louis, Missouri: When I received Doctor Hilding's paper about a week ago, I started to look at as many sections of lungs from cases of asthma and tracheobronchitis as I could find; also because he poses the question as to whether the change, shall we say, of metamorphosis or of degeneration is one that is related to allergy, I looked at some lung sections of animals that we had sensitized to egg albumin and then had treated by putting the egg albumin into their lungs through the nose. I thought that perhaps they might show changes that occur in adult allergy. As a matter of fact, they did not. In none of the sections — and there were some 20 or so rabbits — was this change visible in the early stages of allergy. The changes that we did see were chiefly related to the blood vessels; an increase in the capillary permeability; swelling of the lymphatics around the capillaries; bronchitis and pneumonitis, but no metamorphosis into goblet cells in the epithelium of the bronchi. There was, however, complete destruction of ciliated cells in every case. I could find no cilia except in very occasional places in the bronchial tree.

The cases of tracheobronchitis that we saw showed a destruction of epithelium, just as Doctor Hilding described it. I think his pictures are splendid — exactly as though one were looking through a microscope.

I think one point that his observations bring out in the treatment of these respiratory diseases is the importance of early aid to the patient with bronchoscopy and with tracheotomy when necessary.

I have found it a little difficult to get the internists and the general physicians to agree that bronchoscopy is a very necessary and life-saving procedure. I think Doctor Hilding has shown this quite definitely, and I hope that when his paper is published internists and physicians in general practice will read it and mark it well. We may be able then to prevent some of these deaths from asphyxia.

Another point that Doctor Hilding emphasizes is that thick mucus plugs the bronchi and is anchored by strands of mucus attached to the goblet cells. This brings up a point in the treatment: One must aid the patient to get rid of this mucus by withholding sedatives that will stop the cough reflex and by moistening the mucus as much as possible. To do this one should withold pure oxygen, because it dries the mucus and makes it still more sticky; one should give an oxygen-carbon dioxide mixture and should perform early bronchoscopy and tracheotomy.

THOMAS GALLOWAY, M.D., Evanston, Illinois: How long does it take to develop such a condition as Doctor Hilding has described?

Aspiration in severe asthma may be very valuable, and I am sure that I have saved more than one patient in status asthmaticus by it, but recently I had a very tragic experience with a fine young doctor. For 36 hours he had been in status asthmaticus. Bronchoscopic aspiration was attempted but there was almost no secretion in the larger bronchi nor in the secondary bronchi that could be reached with the fine aspirator.

At autopsy there was very sticky secretion throughout the finer bronchioles. Would irrigation have been of value to remove that secretion along with aspiration? We did use carbon dioxide to liquify the secretions, with apparently some benefit. This patient was kept alive though unconscious with carbon dioxide and oxygen for eight hours but finally succumbed from apparent cerebral anoxia.

A. C. HILDING, M.D., Duluth, Minnesota: I do not know that I can answer Doctor Galloway's question; in fact, I know I cannot. I can simply state the average duration of the asthma in these patients. In the first group, in those with metamorphosis or degeneration, the average duration of the asthma was 11 years. In the second group, characterized by bronchitis, the average was 15 years.

The sections of the rabbit that I showed were taken from one that had been operated upon and the right nostril closed 128 days before it was sacrificed.

The problem of aspiration is a real one. I have also had tragic experiences along that line, trying to get the secretion out and failing to do so. Nevertheless, I think that is the line of attack that we have to take in these patients in extremis and then, of course, we must prevent other patients from reaching that stage if we can.

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Committee Activities

American Committee on Optics and Visual Physiology

The American Committee on Optics and Visual Physiology was originally named the American Committee on Optometry. The early work of the committee consisted in attempting to eliminate the difficulties existing between ophthalmologists and optometrists.

The committee is sponsored by the three national ophthalmological societies, namely, the Section on Ophthalmology of the American Medical Association, the American Ophthalmological Society and the American Academy of Ophthalmology and Otolaryngology. In 1929 its name was changed to the present title.

The committee functions through subcommittees. Each problem presented is referred to a subcommittee which reports to the entire committee at the next meeting.

Some of the subjects under consideration are:

Visual acuity charts
Illumination standards
Motor anomalies and orthoptic training
Classification and nomenclature of motor anomalies
Polarizing lenses
Grading of lenses
Relationship between ophthalmologists and opticians
Specifications of type and paper for books, newspapers and magazines
Revision of optotypes
Screening tests for the examination of eye defects in school children
Visual standards for men in the armed services
Minimal standards of visual acuity for the army air service

The committee cooperates with the Council on Physical Therapy of the American Medical Association in evaluating new ophthalmic instruments.

CONRAD BERENS, M.D., *Secretary*

Industrial Ophthalmology

The Joint Committee of Industrial Ophthalmology has, through its chairman, Dr. A. C. Snell, officially appointed a sub-committee to work on the problem of epidemic keratoconjunctivitis. This sub-committee was appointed because of the considerable incidence of cases of epidemic keratoconjunctivitis in the middle west and because of the un-

usual interest of virologists, epidemiologists and serologists of Illinois and Indiana. The fact that only a meager amount of fully organized research has been undertaken, where rigid controls, as to the criteria for diagnosis, epidemiological history, serology, bacteriology, virological findings and the procedures of treatment exist, seems to make such a study both opportune and imperative at this time. Dr. Murray Sanders has expressed his interest in the idea as developed to date and has offered his fullest cooperation in any phase of the research project.

The sub-committee appointed by Doctor Snell consists of six ophthalmologists and three technical members. The ophthalmologists are:

Dr. Thomas Allen, Chicago, Illinois (also a member of the Joint Committee).

Dr. Harry S. Gradle, representing the University of Illinois Medical School.

Dr. Sanford R. Gifford, representing Northwestern University Medical School.

Dr. Glen Harrison, Beasley Clinic, Waukegan, Illinois.

Dr. H. Woodruff, Joliet, Illinois (consultant of the Kankakee Ordnance Plant, where there are many cases).

In addition to the above mentioned ophthalmologists, the following were appointed:

Dr. Edward A. Piszczek, Epidemiologist of the Illinois State Department of Health.

Dr. H. J. Shaughnessy, Virologist of the Illinois State Department of Health.

Dr. S. O. Levinson, Serologist; Director of the Serum Institute of Michael Reese Hospital.

The first meeting of this sub-committee was held on April 29, 1943, in Doctor Gradle's office. The following criteria were drawn up as being necessary for the diagnosis of a case of epidemic keratoconjunctivitis as included in this study by the six ophthalmologists of the sub-committee:

1. Edematous conjunctivitis.
2. Serous secretion.
3. Preauricular or cervical glands.
4. Monocytic cells in the conjunctival scrapings.

It was agreed that there were probably two stages: (1) an acute stage in which the above conditions were present and in which some cases also may show a membrane; (2) the corneal lesions were considered as indicative of a later stage, almost a complication, and were present in only a certain number of cases. The percentages of corneal involvement vary evidently with the virulence of the virus and the geographical distribution in the country.

Regular clinical and laboratory forms are being drawn up so that the identical procedure in the keeping of records, laboratory work, etc., pertaining to the cases seen by the committee ophthalmologists, will be followed.

HEDWIG S. KUHN, M.D., *Secretary*
Joint Committee of
Industrial Ophthalmology

Age as a Factor in Near Vision Problems of Industrial Production

The importance of any particular group of visual elements in the over-all picture of a nation, continuously tightening the intensity of its industrial war effort, is governed by many factors. Of these factors one only will be discussed —that of age.

In the early months of 1940 when Industry was designated as Defense Industry, the focus of attention, as far as vision was concerned, was the detection and improvement of the defects found *within* Industry. As Defense Industry became War Industry, new and intricate production processes were gradually de-

veloped requiring specialized skills. Training schools were set up for high school age boys and girls where the period of learning had to be constantly shortened. Visual aptitudes were at that time recognized as being of importance in selecting candidates for these schools. The production drive gathered momentum at about the same time that Selective Service went into high gear. The accelerated drafting of young men pulled trained workers out of Industry by the hundreds. Management then had to take on totally green applicants, young women and older men, and had to train them in multiple ways—to operate intricate machines, to man long assembly lines and to inspect an ever increasing volume of special steel, guns, ammunition, armor plate, gears, tanks, etc. This new civilian-labor pool included many who had never worked in industry before, many who had never worked at all and many who had retired from work. The first two groups by and large had paid small attention to their eyes. The grocery clerks, salesmen, domestic servants, barbers, musicians, bookkeepers, housewives, etc., had not been asked to pass any eye tests. Industry discovered very soon that there were many eye defects among them. The older group, emerging from retirement, had, as a matter of course, higher incidence of visual faults. Actually, percentages of the visual defects among the applicants of 1940 and 1943 speak for themselves.

	% 1940	% 1943
Unaided Acuity (naked eye)	18	34*
Working Acuity (wearing whatever correction individual had on)	14	23
Stereopsis	18	26
Color Appreciation	4	5
Muscle Balance	16	23

A study has been made recently of a war industry which is in processes of doubling its payroll as rapidly as it can find men and women able to meet its fairly high minimum visual standards. Among other interesting findings (to be reported shortly in the Journal of the A.M.A.), the spotlight was turned on the striking significance of age as a factor directly related to visual defects in the war production effort. As far as norms for *distance* are concerned, visual defects in individuals in the upper age brackets are more often due to pathology than to refractive errors. However, the picture is entirely changed in an evaluation of *near work* visual skills. This is to be expected, of course, BUT expecting it does not of itself solve the many problems presented. It is here that the responsibilities of the ophthalmologist are immediate and urgent. It is necessary both to search for incipient and existing trouble *within* industry, if acting in the capacity of consultant, and also to search for incipient and existing trouble in one's private practice. Mrs. Smith has a record in our office files stating that she is Mr. William Smith's wife, but—she has *now* a clock number herself and is working in a munitions plant. Johnny Brown, age 15, is the son of Mr. and Mrs. Brown, according to our records, but he too is *now* working on an assembly line. Old Mr. Jones, whose record reads "owns a business—retired" is *now* an inspector in an aircraft plant. They have discovered aches and pains while doing meticulous close work 8-10 hours a day—things get foggy at times and especially so for old Mr. Jones. Right here we find the key to *our* war contribution.

It is up to *us* to make it possible for Mr. Jones to read his micrometer at 8″-10″ from his eyes—not only to read his newspaper at 18″-20″. It is up to *us* to watch for fundus pathology, lens changes, contracted fields and to conserve to the utmost the vision of each individual who comes to our office for whatever reason. It is not enough only to remove the bothersome chalazion

* Almost doubled.

which was the patient's complaint, when he came in for attention. We must also at *that* moment find any existing pathology or any uncorrected refractive error—in other words do a complete examination no matter how trivial the reason the patient gives for his or her visit.

In order to take this discussion out of the realm of the hypothetical, it is necessary only to study the significance of the two graphs included in the text. The relationship between the five cardinal visual functions in employees over 40 years of age and under 40 years of age are indicated in Graph No. 1. In *each* instance the percentage of defects is higher in those *over* 40. Most striking is the

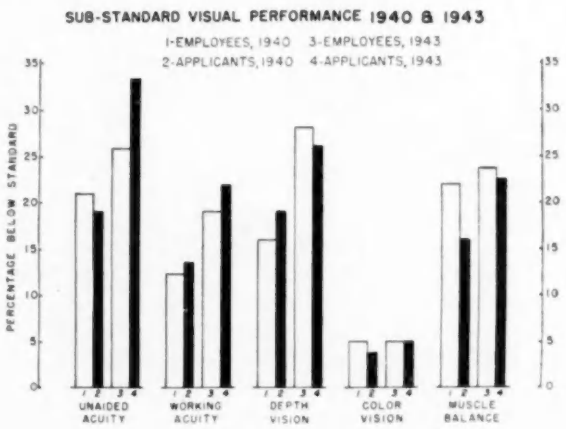

FIG. 1

Without exception the percentage of defects of each type of visual skill is found to be higher in those over 40 years old than among those under 40. The figures of muscle balance have been broken down into near and far, in order to demonstrate the presence of increased problems of near work which have hitherto been neglected.

increase from 20 per cent to 38 per cent in the acuity defects of the unaided eyes (naked eyes) but also of considerable significance is the pattern of muscle imbalances for *near*—this being increased from 10 per cent to 24 per cent as the 40-year age level was stepped over.

The increase in difficulty in color appreciation with age again bears out the previously reported findings of Professor Joseph Tiffin (reported jointly with the author in the November 1942 issue of the Archives of Ophthalmology). The phorias (the increase being almost entirely in the direction of an exophoria) present a real problem to the refractionist as he usually must give a maximum of plus for such close and fixed work distances as 8, 10 and 12 inches. For instance, both perfect acuity and normal near muscle balance have been found to be essential for inspectors. Abnormalities of the latter can cancel out, in great part, the possible perfection of the former. This is especially true when the element of fatigue enters towards the end of the work day.

Studying the differences in *near acuity* with age, as presented in Graph No. 2, we discover again where our point of attention should be. The graph illustrates the percentage of employees over 40 years old and those under 40 years old who fail at different levels of the near acuity test with glasses if customarily worn, namely, *as they were actually working.* At the number 10 level (which is normal, or 20/20 at 13″)

only 12 per cent of these *under* 40 failed while 50 per cent of those *over* 40 failed at this level (or four times as many). The greatest difference in the percentage failing lies in the 7-11 range of vision (which is normal or slightly subnormal). Past that point the demands of ordinary home life bring correction. The employee can no longer see the numbers in the telephone book or the newspaper. The solid black line, representing the older men, *can, must* be, and *is* being brought down to coincide with the dotted line. This is, of course, done by giving these workers their proper near vison correction—usually a simple presbyopic correction but often for an unusual work distance.

It was thought best to focus attention rather sharply in this paper on one problem only (out of the many possible ones), namely, that of visual defects as related to age. Those of us who fight on the home front feel a great urge to seek out the essentials; to find the trouble spots *most* needing attention, and to give our strength, time and best knowledge to solving these "first things first." Our private practice now includes few who can be called people of leisure; it has become a practice of workers—executives at desks or books for 14 hours a day; clerks workng a daily overtime and a six-day week; operators of every known type of machine and assembly linemen; engineers pouring over blueprints. The eyes of the Nation are at work! The *young* eyes of our nation's men are at work in the bombadier's bay, at watch on the bridge of a destroyer, on guard on the desert; the *old* eyes of our nation's men are at work on the home front. Perhaps we can serve these soldiers of industry more effectively as we continue to learn of their special problems and seek the answers.

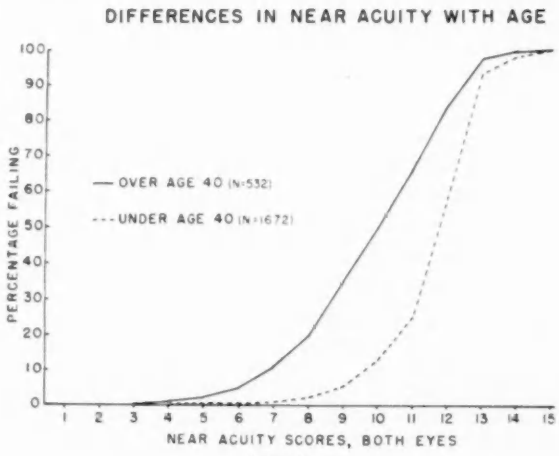

FIG. 2

VISUAL DEFECTS OF EMPLOYEES OVER AND UNDER 40 YEARS OF AGE

Here the graph shows the percentage of employees over forty years old and those under forty years old who fail at different levels of the near acuity test with glasses if customarily worn — *namely as they are now working.*

HEDWIG S. KUHN, M.D., *Secretary*
Joint Committee of
Industrial Ophthalmology

Hotel reservations for the Academy Convention Oct. 10-13, Palmer House, Chicago, should be made well in advance. Be sure to get return railway ticket.

Society Proceedings

AMERICAN OPHTHALMOLOGICAL SOCIETY

The Seventy-Eighth Annual Meeting
June 1, 2, 3, 1942
Hot Springs, Virginia

ALLEN GREENWOOD, M.D., President
Boston, Mass.
EUGENE M. BLAKE, M.D., Secretary
New Haven, Conn.

George Berkeley and "An Essay Towards a New Theory of Vision." Dr. Burton Chance, Philadelphia, Pa.

After assurance that the retina is the essential element, visual studies were concerned chiefly with the refraction of the eye. At the opening of the 18th Century, philosophers were aroused by the publication, in 1709, of "An Essay Towards a New Theory of Vision" by George Berkeley, of Dublin, Ireland, then about 25 years of age. He sought to show "the manner wherein we perceive by Sight, the distance, magnitude and situation of objects," and "the ideas of sight and touch." He declared "the writers of optics had proceeded on wrong principles."

A New Cross-Cylinder Test of Astigmatic Axis, Without Use of Test Type.
Dr. William H. Crisp, Denver, Colo.

All tests, whether subjective or objective, for astigmatism prove at times inconclusive.

Any supposed infallibility of retinoscopy is vitiated by the varying refraction of different parts of the pupil and the impossibility of knowing exactly how large an area of the pupil is habitually utilized for sharp central vision. The retinoscopic finding is not final and requires revision at the trial case in almost all patients. The cross-cylinder test for axis is more completely dependable than astigmatic dials designed for indication of the axis.

In some cases it seems that the patient's judgment may be biased by the influence of individual shapes of letters. Beach's Maltese-cross test aims to meet this difficulty. In the effort to obtain an additional check in such cases, the author has found it effective to make use of the distortions produced in such an astigmatic-axis dial as that of Lancaster and Regan or that of Verhoeff by using the manipulation of the cross cylinder which is commonly applied to the letter chart in testing the axis.

Discussion

Dr. Walter B. Lancaster, Dartmouth, N. H.: I cannot let an opportunity pass to express my appreciation of what I call a masterly presentation of how we should handle the problem of measuring refractive errors. I hope you will all study this carefully. I refer here to the methods rather than to the results. We all have our own special way of applying these principles. I want to mention only one. The speaker said that astigmatic dials frequently give misleading and unsatisfactory clues as to the axis, and any one who has used them knows that is so. I have found that one of the best ways of arriving at a more definite clue is the proper selection of the amount of fogging. You cannot use the astigmatic dials successfully if you allow accommodation to run rampant. You must use a strong enough positive lens to fog the patient. You will find that, if you vary the amount of that fogging a little more or a little less, you get different axes in a few patients, and the question is, what axis should you choose? Suppose you conclude from various tests — including the record of previous glasses — that the true correction is approximately +2 sph. ⊃ —0.75 cyl., and you wish to get a definite axis. If you use a plus 2.25, you will get one axis. If you use a plus 1.75, you get, in a few cases, a different axis. The proper strength sphere to use for the fogging for the exact axis is the sphere which puts one end of the conoid of Sturm exactly on the retina, not a little in front or a little behind. You cannot do that with certainty at the beginning, because you do not know the value of the sphere and the cylinder, but toward the end of the test, when you do know these values to a close approximation, perhaps after measuring the sphere with both eyes and using the approximate cylinder correction, then make a final test for the axis with the sphere, which equals the total correction

in the strongest meridian. In the case just described (+2 sph. ⊃ —0.75 cyl.) the total sphere in the strongest meridian is +2. Suppose it were not expressed as a minus cylinder, but as a plus (+1.50 sph. +0.75 cyl.). The total sphere in the strongest meridian is now 1.50 +0.75 = 2.25. Then what should you use? Add these together and you get 2.25. That is the sphere which brings you on the exact end of the conoid of Sturm. If it is a minus cylinder, you use the sphere as it is. If it is a plus with a plus, you add them together, and that gives you the proper fogging for the exact axis. With that method you will have somewhat fewer confusing findings.

Dr. F. H. Verhoeff, Boston, Mass.: I have always relied upon astigmatic dials as a means of arriving at a correct result. Of course, we all believe, although it may not be strictly true, that an astigmatic correction that is best for reading test letters is the best for all other purposes. The final test of astigmatism, therefore, is that of the ability to read test letters. Astigmatic charts, cross cylinders, etc., provide a simple and rapid way of reaching the last stages of the final test. As a rule, I find I cannot improve, by means of test letters, the correction I quickly obtain by the use of astigmatic charts. If one relies upon test letters alone, the patient becomes so fatigued that the results are inconsistent. My main reason for speaking now is to call attention to the fact that on my astigmatic charts the lines have never been as solidly black, and the paper upon which they are printed has never been as white as it should be. These charts were first printed over forty years ago, and I believe are still made from the original plates. A photograph of my chart and one of another type in which the lines are much blacker and the paper whiter, therefore, do not provide a fair test of the value of the fine lines on my chart. The comparison should be made with the same kind of paper and the same blackness of the lines in both charts. My charts, as furnished by the manufacturer, can be greatly improved by carefully blackening the lines with India ink.

Dr. Edward Jackson, Denver, Colo.: The astigmatic chart is, as has been said, an approximate approach toward the meridians. An approach that is more nearly final in that it simplifies the problem of the observer — the patient is making the observation — is to reduce the astigmatic chart to its simplest form of three lines in one direction and three lines in the other. This sort of a chart is to be rotated in any direction, and in using such test objects with the cross cylinder I have found this: the three lines at right angles to one another is the best means of comparing the different meridians.

Dr. Frank P. Smart, Norfolk, Va.: There are two points I want to mention. The first is that in the original paper all the illustrations had the picture of the dial placed above and in front of the test letters, leaving the twentieth or fifteenth line of letters exposed. It seemed to me that the differences were just as apparent in the test letters as on the dial face.

The second point is one in which I have to differ from Dr. Lancaster. I have found that whenever one gets the circle of least confusion off the retina, bizarre results ensue. If, instead of fogging, one keeps the circle of least confusion on the retina at all times, the most positive, rapid and accurate results are obtained from the use of cross cylinders, both as to the amount and as to the axis of the cylindric correction.

Dr. Conrad Berens, New York, N. Y.: I would like to ask whether there has been any question of including the smaller Beach test objects in these charts. When Beach brought out these test letters and characters, they were so large that I could not obtain very good results. Later we used the "Z" and "N" and the Maltese cross in different sizes to conform to visual acuity as good as 20/10. Using the Beach characters in that way I found them accurate and apparently more useful in my hands for the final determination of the astigmatic axis than the astigmatic dials. Has Dr. Crisp had the circles reduced in size so that he can then compare his astigmatic dials with the Beach test objects, which have been reduced in size on a chart printed by the American Optical Company?

Dr. William H. Crisp, closing: Time hardly allowed for the discussion of some points I should have liked to mention. Because the letter chart was in place, and also the Maltese cross which I printed myself in India ink and cemented on with rubber cement, I left them so that you could compare them. The camera does not record irregular astigmatism, and we

are going to use these tests on patients who do have irregular astigmatism, and to this extent the photographs may not tell quite the whole story as to the effect upon the patient of the cross-cylinder combination with the letters, the Maltese cross, and the astigmatic dial respectively.

As regards fogging, I find that having the patient appreciably fogged at times helps in the earlier stages, but my impression is that toward the end of my examination the patients get the best results if I have their accommodation relaxed without definite fogging.

Dr. Lancaster spoke about the conoid of Sturm. When we get down to the point at which I want the greatest refinement in testing, we have almost no conoid of Sturm.

Correction of Low Astigmia. Dr. S. Judd Beach, Portland, Maine

There is no agreement about either the importance of cylindric errors or the best tests for them. Discussion has been too much influenced by the preferences of the examiner rather than the peculiarities of the patient. A variant of standard technics is described, requiring no special equipment, and adapted to many obscure cases.

Discussion

Dr. John Green, St. Louis, Mo.: There is a wide variation of opinion as to the effect of uncorrected low errors of astigmatism on ocular comfort and general well-being. Some cannot bring themselves to believe that low astigmatic errors give rise to symptoms; hence they disregard them altogether or are content with an approximation as to both amount and axis. To many patients these approximations are satisfying. It is my contention that the examiner should have sufficient pride in his work to endeavor to perform a precision job on every one of his patients.

It is my practice to correct fully high astigmatism. But there are exceptions. When the prior correction has been a sphere or a sphero-cylinder, it seems best at first to undercorrect the astigmatism, lest the patient, because of the distortion of objects or a sense of dwarfism or gigantism, discontinues the use of the glasses.

I agree with Dr. Beach that "patients differ completely in their reaction to the same amount of astigmatism." It is perhaps unimportant to insist on the constant wearing of low cylinders of equal strength and axis. On the other hand, it seems advisable to prescribe for constant wear astigmatic errors under the following four conditions:

1. When the astigmatism is equal but at different axes.
2. Where there is a difference in the degree of astigmatism.
3. When the astigmatism differs in degree and is also at different axes.
4. When there is myopic astigmatism in one eye and hyperopic in the other.

Dr. Beach states that, as the initial step, he places in the trial frame the correction as estimated by retinoscopy; and this correction is made the basis for the subsequent procedure. Personally, I would prefer a somewhat closer approximation by carrying out the familiar "round of the trial case," as advocated by Duane, using plus and minus 0.25 spheres and cylinders. It may be inferred— and correctly — that I am quite suspicious of the findings of the retinoscope and prefer to have these corrected or confirmed by subjective tests.

Dr. Beach points out that a patient resents additional fogging, which is implicit in the standard technic of adding plus cylinders either in the axis of the trial-frame cylinder or at right angles to it. With this technic the sphere, being slightly overcorrected and the cylinder presumably of the proper strength, the patient is disconcerted to find that the next step is the placement of an auxiliary lens which deprives him of his 6/10 or 6/7.5 line of letters. He cannot understand why he is suddenly thrust back into a fog. To forestall the patient's unfavorable comments, I frankly admit that the auxiliary lenses produce blur, but point out that it is his job to select the lesser of the two evils. At this point it does no harm to refer casually to the superior intelligence and extraordinary powers of observation of your patient. This enhancement of his ego is helpful in riveting his attention to the matter in hand.

Dr. Beach's method is acceptable to less intelligent patients who are annoyed by the overcorrection of the sphere. Their annoyance will not be increased by the addition of an auxiliary minus cylinder at the improper axis, and they will welcome the increased acuity when the added minus cylinder in the proper axis gives a clear indication as to increase or decrease of cylindric strength.

Dr. Beach offers, by means of subtraction technic, a method of determining the presence or absence of low astigmatism at the four cardinal axes. The inferences which he draws from the several maneuvers described are sound and in accordance with accepted principles of optics. He depends for still more precise placement of the axis on the relative strength of the "signals" indicated by auxiliary minus cylinders. Does he, in practice, prefer this method to the well known and widely appreciated use of the cross cylinder for this purpose?

Assuming that the axis has been accurately determined, and the strength of the cylinder fixed by standard technics, including the cross lines of the Lancaster or Camp-Thomas charts, is it possible to refine the estimation of strength to a still higher degree? I believe that it is. For this purpose I use a 0.06 cross cylinder (first suggested in 1932). This consists of a lens ground with a minus 0.06 D. sphere on one side and a plus 0.12 D. cylinder on the other. Let us assume a low hyperopic astigmatism, axis 90, which has been perfectly corrected. An observant patient will detect no difference in the sharpness of the smallest letters in the two positions of this cylinder. When this cylinder is placed with the plus axis parallel to the plus axis in the trial frame, the horizontal lines will appear sharper. With the minus axis thus placed the vertical lines will be better defined.

When using the 0.06 cross cylinder there must be no overcorrection of the sphere.

Rocking the cylinder, especially in low astigmatism, often fails to give any positive indication. In higher astigmatisms it is very useful, especially if the patient is permitted to do the rocking. A keen individual can be incited to utmost effort by thus physically participating in the determination of his error. He is thus encouraged to work out his own salvation in partnership with the examiner.

It is indeed a pity that many examiners are wedded to a single method. In the determination of low astigmatism the examiner should have at his command the various technics, and should modify his methods in conformity with the mental and psychic peculiarities of his patient. Particularly is the neglect of astigmatic charts hard to understand. Granted that the dial is occasionally misleading, it is in most cases of the utmost value in determining the presence, the approximate axis and the strength of the correcting cylinder. It is remarkable and deplorable that very few refraction clinics in this country are equipped with astigmatic charts.

I am indebted to Dr. Beach for presenting a refinement of refraction technic that is proving increasingly satisfying in practice.

Dr. William H. Crisp, Denver, Colo.: Dr. Beach has very properly called attention to the fact that, in dealing with a patient who is sensitive to a low error of astigmatism, it is rather easy to overlook an off axis with low amount of astigmatism. In attempting to trace these fractions, where other methods so far have not shown the presence of astigmatism, I use one of two methods, and I am apt to check one by the other. I should say that, instead of using four principal meridians to check, I systematically use six, and instead of taking 90° and 180°, and 45° and 135,° I take 90° and 180°, 30° and 120°, and 60° and 150°. I take a 0.12 D. cylinder, either plus or minus, and I hold it for example in the vertical or the horizontal position alternately, and over this simple cylinder I use a 0.12 D. or sometimes a 0.25 D. cross cylinder. The patient is asked to say whether the two lines of the single cross are more equal in one position of the cross cylinder or in the other. If I am adding plus to his plus vertically and he does not need more plus vertically, he will say the lines are more equal with the minus of the cross cylinder placed vertically. If in each of the six meridians he rejects even 0.12 D. of cylinder, I may assume that there is no correctible astigmatic error.

I can do the same thing with the cross cylinder and the letters, running through the six meridians one after the other. If in this way I once get a start on a low astigmatic error, I can put in the frame a 0.12 or 0.25 cylinder and then proceed to test the axis. In that way it is often possible to pick up a low error at an oblique axis that could not have been picked up in any other way.

Dr. Burton Chance, Philadelphia, Pa.: Dr. Lancaster, as he met me last night, asked did I deem it our duty to make patients happy, to which I replied that I did. While listening to the essayist I thought that if there were a guest here who has

been debating as to whether or not ophthalmology should be taken up, he might suddenly disappear because he had begun to doubt that he could ever be able to prescribe a glass with which the patient would be happy. Reference has been made to the consideration of the eye as a faulty optical instrument. Helmholtz has been charged with having made the statement that the eye is a faulty instrument. In truth he did not so state; what he did say was that if one were to be offered an instrument having the optical defects inherent in the human eye, it would never be accepted from the instrument maker. Our eyes are organs capable of making their own adjustments, and it is impossible to treat them in the way we would operate a fixed instrument.

With reference to the necessity to correct low errors: In perusing the early records of the late Dr. William Thomson, I have found that many a patient had been sent to him by Weir Mitchell because of minor nervous troubles. Not a few of his prescriptions, which both Thomson and Mitchell carried out with the greatest particularity, call for much less than one diopter. The letters sent by the patients to Thomson state that they had been made happy by their spectacles, with which they had been relieved of pains and other troubles not previously remedied.

Dr. C. A. Clapp, Baltimore, Md.: I would like to ask Dr. Beach one question: When I prescribe a 25 cylinder and the patient comes back with relief of headache and other symptoms, am I practicing ophthalmology or psychiatry?

Dr. F. H. Verhoeff, Boston, Mass.: I presume that Dr. Beach carries out these procedures under cycloplegia, and then uses them again after the cycloplegia has worn off. I should like to ask him whether he gets different results in the two sets of tests, and if so, which he relies upon.

Dr. Nelson M. Black, Miami, Fla.: I should like to ask Dr. Beach, in correcting these low degrees of astigmatism, whether he applies the same method in presbyopic cases.

Dr. Alfred Cowan, Philadelphia, Pa.: The thing I like about Dr. Beach's test is that he utilizes the ordinary test-case lenses. There is no such thing as a human eye that is as mathematically exact as a schematic eye, and lenses which are given to a patient for the correction of a certain error are for the purpose of giving this patient comfort and relief. It is a different thing to prescribe glasses for a person whose vision is only 6/12 than for one, for instance, with 6/5 vision. The one with 6/5 vision will demand a much more nearly exact correction than the one with 6/12 vision. In a subjective test it is important that the correction be aproximate before refinements are attempted. The best refractionist is the one who not only arrives at the proper conclusion, but who does so rapidly. For instance, in doing a retinoscopy it is wrong to start out with 0.50 diopter differences. Rather overcorrect first then undercorrect, and in this way the work will be done within one diopter. Not until then should fine differences be studied. It is up to the ophthalmologist to decide which test or tests are applicable to a particular patient. If all tests were used on each person, we would have to work with him three or four days. Dr. Beach brings out a test used with ordinary trial-case lenses, and I think it is the proper procedure to utilize the resources of the ordinary trial case before any of the methods of refinement are attempted. The quicker we arrive at a near-refraction, the better the refraction will be done and the better the patient will be satisfied.

Dr. S. Judd Beach, closing: The catholicity of the speakers has pleased me. I expected more prejudice in favor of particular tests. I appreciate what Dr. Green and Dr. Crisp said about various little modifications to improve this one. Very likely Dr. Crisp's use of six axes instead of four might be adapted here in some cases to great advantage.

With regard to what Dr. Green said about the use of the cross cylinder, I am quite aware of all its merits and use it constantly. Just why it fails at times I do not know. Possibly changing the cylinder before the patients' eyes may disconcert them. There are probably other reasons. We know that all the tests have their drawbacks.

In answer to Dr. Clapp, I think that Dr. Lancaster's remark that we cannot divorce psychiatry and ophthalmology is the best answer.

In reply to Dr. Verhoeff, I suppose I shall differ with a large proportion of the practicing ophthalmologists in relying on

the post-cycloplegic examination when it differs from the cycloplegic.

I should answer "yes" to Dr. Black's question, although I do not carry through to the prescription.

Until this year I agreed perfectly with Dr. Cowan's belief that you can ignore the refinements in an eye with low vision. In general, tests not quite so refined in the examination of amblyopic eyes have been satisfactory, but I have just had two patients with vision around 6/30 who are highly sensitive to cylindric correction.

Practical Importance of Aniseikonia. Dr. Edward Jackson, Denver, Colorado

The nature and importance of aniseikonia is described and illustrated by three extreme cases. One was the case of Sir George B. Airy, reported in 1827, and two others. The latter cases following the extraction of cataract from one eye, while the other eye still retained useful vision. These cases indicate the possibility of new visual coordinations. These may be formed even late in life.

A Reply to Criticisms of Aniseikonia. Dr. Walter B. Lancaster, Hanover, New Hampshire

Heterophoria, especially anisophoria, as a source of error.

Peripheral fusion, which is depended on to measure aniseikonia with the eikonometer, is unreliable in the presence of central images which, though dissimilar, have a tendency to become fused or aligned.

Findings with frontal plane horopter and with grid-nonius horopter.

Enormous size differences that occur in the normal use of the eyes, e.g., asymmetric convergence.

In using the eyes on small objects, differences in size would be below the threshold of vision and yet use of the eyes for near is said to be more often a cause of symptoms than use for distance.

Discussion

Dr. H. Burian, Hanover, N. H. (by invitation): In connection with Dr. Jackson's paper, I should like to mention the following: It is quite true that we may get accustomed to the wearing of glasses which at first may cause certain distortions of space, and one might well assume once one gets accustomed to such distortions the glasses are satisfactory, and no further therapeutic procedures are needed. However, we must consider that the visual act consists of two phases or aspects. One is the acquired learned clues which are represented by the monocular clues of perspective, shade color, and so forth. The other is those clues which are innate and based on incongruity of the ocular images. By wearing different sized lenses of considerable amount — 3, 4, 6, and 8 per cent — for some time over the last two or three years I was able, after a few days, to accustom myself to those glasses in the sense that I no longer saw the desk top tilted or other distortions in space. However, unless I had myself under appropriate conditions, that is to say, whenever in the field of vision there were no perspective clues, the size effect returned. The experiments about which I am talking have been quite strenuous, and have taken up most of my time during the last two or three years, and I am looking forward to publishing them some time, but the main result is that we do get accustomed to certain inequalities in ocular images, but it is only apparent; we do not actually grow accustomed to them, because we cannot change our disparities. What we can change is that part of our visual apparatus which is acquired by experience — which has been learned.

In amplifying some of Dr. Lancaster's statements, I should like to show one slide. In this slide measurements are shown which were taken on 107 flight cadets, 341 college students and 100 clinical patients referred because of aniseikonia. This slide is most revealing. It shows in the flight cadets — in young men who have been very highly selected, in that these cadets were at Pensacola, which is not a primary school — that the amount of aniseikonia actually measured with the eikonometer is very small. Of these, 93 per cent showed an aniseikonia of ½ per cent or less, and there was only one man who had an aniseikonia of 1 per cent. Among the college students, who were healthy young men with relatively few ocular complaints, the amount of aniseikonia found is considerably higher. Of the 341 college students, the percentage of those measuring ½ per cent of aniseikonia or less drops to 33 per cent. The picture is entirely opposite in the clinical patients who came with complaints. In these patients only 15 or 16 per cent showed an aniseikonia of 0.5 per cent or less; the vast majority had an aniseikonia which is higher, and which we consider

of clinical importance. These figures are of interest insofar as they allow conclusions which answer objections raised to certain clinical aspects of aniseikonia. The fact that the majority of the patients referred for an examination showed a much larger amount than is to be found in perfectly normal individuals does in itself justify the endeavor to correct this anomaly, especially if previous attempts to relieve the patient by other methods have failed. These figures also clearly indicate that the favorable results achieved with aniseikonic corrections on the basis of painstaking measurements cannot be explained away by the suggestive influence of the examination or the unusual glasses.

As regards this last point, which I should like to have Dr. Lancaster amplify, there is an even more convincing argument. There are numerous cases which have been reexamined over a period of from five to ten years. In a good many of these cases the amount of aniseikonia varied only within the limits of error of the method. In others there were some changes which were, however, most frequently attributable to changes in the refractive correction needed. Such a stability of the measurements can occur only when a physiologic but not a psychologic factor is determined.

Finally, there is one more point I should like to raise in connection with Dr. Jackson's interesting case of aphakia. Naturally we have been most interested in that at Dartmouth, and have examined a great number of such patients. By putting on refractive corrections, such patients are at times able to have binocular single vision, but that binocular single vision is present only in a very small area around the fixation point if the patients are measured with proper methods for the determination of their binocular vision. It does not cover the whole field of binocular vision. It is most unusual to find any one who does have fusion under those circumstances, but we have always been able to restore binocular single vision over the whole field if their aniseikonia were measured and corrected, except where some other difficulty intervened, as the presence of strabismus. It is important that this be corrected a year or so after the loss of the lens, otherwise the separation that sets in results in a divergent strabismus. We have usually worked with younger persons, because we do not think it worth while to go to the trouble of working with older subjects, who are usually happy without binocular vision.

A second point is that the refractive and aniseikonic correction cannot be combined in single lenses in cases of unilateral aphakia, because of the enormous size difference. The resulting spectacles are unsightly and have poor field qualities, as you can see from this slide. We have, therefore, in such cases resorted to the prescription of contact lenses; by computing the curvatures and the thickness of these lenses, the size difference as well as the refractive error can be corrected. Professor Ogle has reduced this somewhat complicated computation to monographs which can easily be handled by an assistant.

A by-product of our work with contact lenses in cases of unilateral aphakia, as well as other cases of aniseikonia, is the light they throw on the problem of anisophoria as opposed to aniseikonia. With the contact lenses not containing the correction for aniseikonia, the patient measures a certain amount of size difference. If the contact lenses are now properly computed from the aniseikonic data, the patient should show no aniseikonia. There would be no possibility for refractive lenses introducing a differential prismatic effect. Such cases have actually been seen at the Dartmouth Eye Institute and have further strengthened the conviction that a size difference, not an anisophoria, is being measured by the eikonometer.

Dr. Conrad Berens, New York, N. Y.: I shall first say a word about Dr. Jackson's experience. For a good many years I was deeply interested in seeing whether I could add to the comfort of those patients in whom cataract had been removed from one eye, that is, to give them complete or partial satisfaction by developing single binocular vision and good amplitude of fusion. I did obtain binocular vision and some fusion, but, as a rule, they became uncomfortable after a short time and gave up the idea of fusion. One patient, a woman who was anxious because her eye tended to "wander out" somewhat, exercised for three or four months and the orthoptic technician said "you are making a mistake," but the patient wished to continue. This woman persisted and did obtain some fusion, but she has since neglected that eye. I found, by centering the lenses lower, that she was

somewhat benefited. In the last three years I have seen patients in New York and at Dartmouth who have worn contact lenses with iseikonic lenses, but I have found that even this group is not entirely comfortable, so I usually tell patients, before extracting a cataract, if the other eye has good visual acuity that they may be uncomfortable and may continue to be so.

I thought it might be of some interest to compare the studies on 450 patients examined during the last year with the previous study. In the previous study 75 per cent of 502 patients who wore their iseikonic correction reported partial or complete relief from unpleasant symptoms. In the study on these 450 patients seen during 1941 there were incomplete ophthalmo-eikonometer examinations in 32 patients. One of the greatest difficulties encountered is the patient whose visual acuity in one eye is too low to be tested. There was no appreciable aniseikonia in 122 cases. Aniseikonia was present, but no iseikonic prescription was given in 50 cases. Iseikonic prescription was given but not filled in 33 instances. Iseikonic corrections were worn, but there was no report on the results in 29 patients. Iseikonic corrections were worn with no improvement of symptoms in 38 cases. In the remaining three groups in which iseikonic corrections were worn there was slight improvement in 33, moderate improvement in 58 and marked and nearly complete relief in 55. In other words, of the 184 patients who wore iseikonic corrections and reported their results to us, 146 or 79 per cent, claimed they were benefited. In the previous group only 75 per cent claimed they were benefited. I believe this higher percentage of reported improvement may be caused by several factors. First, the examiner for the department of aniseikonia does not recommend correction so often; second, the ophthalmologists referring patients for examination select them more knowingly because of greater experience, and, third, because this report is made of results with a shorter time interval between the time when the lenses were obtained and the questionnaires answered. It has been our experience that some patients seem to be benefited only temporarily and find later that iseikonic lenses may be discarded or ordinary lenses worn with comfort.

Unquestionably many patients can wear ordinary lenses or even wear no lenses and still complain of no symptoms although they seem to have the same refractive error as patients who apparently require iseikonic lenses for relief of annoying asthenopia. Physical, psychologic and ocular factors which make these patients hypersensitive should be investigated and, if possible, eliminated.

Dr. William H. Crisp, Denver, Colo.: I should like to know with regard to Dr. Lancaster and Dr. Burian's work whether the test was made with or without cycloplegia.

Dr. Francis Heed Adler, Philadelphia, Pa.: May I ask Dr. Lancaster and Dr. Burian a question. Will they please show the last slide again? To me it was not very convincing. The group of flight cadets are separated from the group of normal students further than the clinical patients are. The flight cadets, being practically emmetropic, were free from aniseikonia, but the clinical patients who had complaints were very smiliar to the 341 college students who did not, and that to me is not very convincing evidence that the difficulties of the patients are due to aniseikonia.

Dr. J. S. Friedenwald, Baltimore, Md.: Dr. Lancaster has referred to a small piece of work which I did a number of years ago, and I would therefore like to discuss some of the points he has raised.

Dr. Lancaster says that phoria has no bearing on the readings made with the eikonometer. In the early literature from Dartmouth, a great deal of emphasis was placed upon a phenomenon closely related to phoria to which Professor Ames gave the name "retinal slip." It is important to know whether Dr. Lancaster believes that the early work of Professor Ames was erroneous. On the other hand, if the early work on retinal slip is not to be denied, then it follows that a patient whose phoria varies with the direction of gaze will give erroneous readings on the eikonometer. In the study which I reported six years ago it was found that the incidence and degree of "anisophoria" among office patients in Baltimore was approximately equal to the incidence and degree of "aniseikonia" among office patients at Dartmouth. It remains to be proved, therefore, whether or not any of

the patients giving positive readings with the eikonometer really have aniseikonia. The issue could be very simply settled if duplicate measurements with the eikonometer, and with a phorometer at different directions of gaze, would be made on a substantial series of patients.

Dr. Lancaster places a great deal of emphasis on correlation between readings on the eikonometer and those with complex measurements of space judgment. The mechanism of space judgment remains very obscure, and the habitual innervation of the eye muscles to various directions of gaze may play an important role in space judgment. It follows that a person with abnormal ocular motility, for instance, with anisophoria, may make erroneous space judgments detectable with the tests which Dr. Lancaster has reported. An example of the role that innervation sense regarding the direction of gaze plays in space judgment is the fact that on asymmetric convergence, a normal person makes approximately correct space judgments, interpreting the disparity in his retinal images differently on lateral gaze than when he looks directly ahead. Dr. Ames explains this by assuming a physiologic variation in perceived image size with asymmetric convergence movements. No evidence has been presented to substantiate Dr. Ames' hypothesis, but if it is correct, it means that the perceived image size is intimately influenced by the innervation sense of the direction of gaze, which Dr. Lancaster denies.

The unequivocal measurement of aniseikonia is very difficult. Some years ago Dr. Burian, of the Dartmouth staff, developed an instrument in which the patient was not required to make conjugate ocular rotations in order to make a reading. Criticisms of this instrument by Dr. Verhoeff have never been answered. However, analyses of paired readings on the Burian eikonometer and on the ordinary eikonometer have not been reported. If it could be shown that there was a close correlation between the readings with the two instruments, the Dartmouth theory might be strongly supported. If the readings with the two instruments are not generally in accord, one would be entitled to conclude that the ordinary eikonometer generally measures not aniseikonia but anisophoria.

Dr. Robert H. Courtney, Richmond, Va.: Twelve years ago Dr. Cowan drew my attention to the fact that aphakic eyes, with a high degree of astigmatism, could be corrected better if they had a minus cylinder in the posterior chamber of the trial frame, combined with a plus sphere in the anterior chamber. This limits a patient's field of vision considerably, but it gives him a remarkable amount of comfort.

About a year later a patient consulted me because of poor vision in the left eye and headaches for which she had not been able to obtain relief. At that time the patient had normal vision in the right eye and less than 20/200 in the left eye. There was present a conical cornea in the left eye. Her refraction in the right eye was: + 50 cyl. ax. 90°, but in the left eye was + 4.50 sph. ⊃ — 7.50 cyl., ax. 95°, which gave her normal vision. Doubting her ability to wear this correction, I asked her to stay in the office for a day's trial of these lenses. The patient wore them with perfect comfort. During the next few days I refined the correction according to Dr. Cowan's suggestion, and this proved to be more satisfactory. There has been a gradual increase in the total correction in the left eye during the last ten years.

The patient has developed a conical cornea in the right eye, which appears to be developing as rapidly as it had in the left eye. In spite of the high degree of astigmatism, the patient does her school work with perfect comfort for the better part of each year. Whether a contact glass would have prevented an increase of the conical cornea I do not know. The patient was well satisfied with the correction of her astigmatism in spectacles, and did not desire the examination for contact lenses. Within the last two years this patient has developed ruptures of Descemet's membrane, and the vision in the left eye cannot be improved to normal.

Dr. Burton Chance, Philadelphia, Pa.: I wish to remind Dr. Jackson that there was an earlier case than that recorded by Airy. Pepys, the famous diarist, had much trouble with his eyes. It was believed that this was caused by the writing of his diary, but certain symptoms of which he complained were such as we would now say were due to inequalities in the fusion by the two eyes. At one of his parties there were a number of medical men. Pepys asked one of them to explain whether it was really true that we see with only one eye, to which he re-

ceived the reply that we see with two eyes which act together. But Pepys was not able to see equally well with his two eyes, and as presbyopia had come on, he could write only with a great deal of pain, so he ceased keeping his diary.

Dr. Edward Jackson, closing: To discuss the discussion, I believe that the members listening would all get the impression that we do not know much of this work, and the most important factor in vision we know nothing about—the development of the neural and cerebral coordination and anatomy; even physics will not help up with that. Such a patient as has just been mentioned by Dr. Chance was right-eyed and did not know about it, indicating the importance of the psychology of vision, and I feel that we all have impressions about it, but we really have very little that can be called scientific observations on the psychology of vision.

Dr. Walter B. Lancaster, closing: In reply to Dr. Crisp's question, I might say we have on our staff a man trained at Wills Eye Hospital; does that answer your question whether we use cycloplegia? He uses it ten times as often as I do.

It is apparent that Mr. Ames feels convinced of the soundness of his position—that the eikonometer measures aniseikonia, whereas, on the other hand, Dr. Friedenwald is not convinced that this is proved, but thinks anisophoria is the source of the erroneous conclusions. My own position is this: Mr. Ames' claim that the standard eikonometer does measure aniseikonia is fully substantiated by a mass of evidence only a small part of which has been published. I have repeated many of the experiments myself. I have made tests on an apparatus which I devised to measure aniseikonia without the possibility of anisophoria entering into the problem since the gaze is continuously in the primary position, with no shifting. This confirms me in my belief. It also answers Dr. Friedenwald's extraordinary statement: "It remains to be proved, therefore, whether or not any of the patients giving positive readings with the eikonometer really have aniseikonia."

The confirmation is a double one: it shows agreement in the measurements on the two types of eikonometer and it shows that putting on aniseikonic lenses corrects the aniseikonia as measured on both instruments.

Moreover, I have measured patients for anisophoria with this and other devices. This apparatus was provided with small lights ("muscle lights"), one central and one 20° from the center to measure the phoria in the primary position, and 20° right, left, up, down or oblique, thus measuring the anisophoria. I found anisophoria was almost universal (as is aniseikonia). As stated in my paper, I regard a certain amount of anisophoria as natural and normal — just what one would expect to find.

In measuring aniseikonia on the standard eikonometer, heterophoria may be very troublesome if it is not corrected. It is not true that "it has no bearing on the readings made with the eikonometer," and if I said or implied that, I was wrong. One of the cases reported in the paper illustrates this very well. The magnitude of the aniseikonia as measured with and without correction of the phoria (usually by prisms) will differ materially.

In short, my conviction is that there is ample evidence for the existence and importance of aniseikonia, and that the standard eikonometer makes valid measurements confirmed by the space eikonometer and numerous other crucial tests. On the other hand, I am convinced that anisophoria is very common, but not usually a source of trouble, probably because so small a part of our seeing is done outside of the central area with a diameter of 15° within which anisophoria is markedly less than outside of it.

The magnitudes being measured by the eikonometer when compared with those measured by the phorometer are very significant. A size difference of 1 per cent means 1 per cent of the distance from the center target to the dash 4° away — 1 per cent of 4° is 1 per cent of 240′ equals 2.4′. A letter on the 6/6 line or 20/20 line subtends 5′.

The difference which the patient has to detect in being measured for 1 per cent aniseikonia is 2.4′, or less than half the size of the 6/6 letters. That is why only persons with good visual acuity can be examined on the eikonometer and why poor observers are unsatisfactory patients.

Phorias, on the other hand, are usually measured in prism-diopters (Dr. Friedenwald used prism degrees): 1△ equals about ½° or 30′. Compare this with 2.4′. In measuring phorias the unit is 13 times as large as in measuring aniseikonia. All

clinicians who have had experience with the eikonometer know that heterophoria affects the reading of the eikonometer, but that anisophoria is what is being measured is easily disproved. Moreover, aniseikonia readily explains certain phenomena of space perception which are not explainable without it, and which are definitely not explainable on the basis of anisophoria.

Dr. Friedenwald makes light of the evidence from the space eikonometer which is in favor of findings with the standard eikonometer. Independent evidence based on data derived from entirely different and therefore independent methods of investigation seems to me the very strongest and most convincing kind of evidence. The ground on which Dr. Friedenwald discounts this evidence is particularly weak. He says: "The mechanism of space judgment remains very obscure, and the habitual innervation of the eye muscles to various deviations of gaze may play an important role in space judgment." This is a surmise on Dr. Friedenwald's part, with very little basis of fact. When I look over here to the right with my left eye covered (illustrating), if my right lateral rectus muscle is paralyzed, I locate the point over there much farther to the right than it really is. I am guided by my innervational clues, and there is no question about the importance of innervational clues in such cases, but they do not suffice for the fine points of localization in normal eyes, and it is easy to cite cases where they are even misleading. Thus a prism base will make us diverge our eyes. Innervationally divergence is associated with greater distance, yet objects usually appear nearer not farther with such prisms — a puzzle elucidated by Ames.

Surgery of the Inferior Oblique at or Near the Insertion. Dr. James Watson White, New York, N. Y.

Surgery of the inferior oblique when done at the origin proved to lack modifications. A complete tenotomy gave in some cases the desired result, while in others there was an undercorrection. This led to a myomectomy in which a portion of the muscle from 5 to 10 mm. was resected. This gave more uniform results when a complete paralysis was desired. Attempts were made to get a partial effect by opening the muscle sheath and excising a portion of the muscle fibers. Later, incisions were made on either side of the muscle as a partial tenotomy. These proved unsatisfactory.

In 1936, I receded the muscle at or near the insertion. This proved so satisfactory that it has been continued with increasing accuracy and confidence. It is a cite of choice when resecting or receding an external rectus, when the convergent or divergent strabismus is associated with an overacting inferior oblique of the eye to be operated upon. A tuck at this cite following Wheeler's technic for tucking the superior oblique has given less satisfactory results, but is preferred to the advancement over the margin of the orbit.

Discussion

Dr. John H. Dunnington, New York, N. Y.: It is always a pleasure to hear Dr. White expound on ocular muscle anomalies, for his advice is sound and his judgment is tempered by the wisdom of experience. The operation of recession of the inferior oblique has intrigued me for two reasons: (1) Tenotomy of the inferior oblique at its insertion has not been a satisfactory procedure; (2) Myectomy of the inferior oblique at its origin has produced inconsistent results.

I cannot agree with his statement that these two procedures produce similar results. In my hands tenotomy of the inferior oblique at its insertion has caused almost invariably a complete paralysis of the muscle, paralleling in every way those rare cases of isolated congenital paralysis of the inferior oblique. On the other hand, myectomy of the inferior oblique at its origin has not abolished all upward movement in the nasal field — in fact, in some instances an upshoot has persisted in spite of the excision of 5 mm. of the muscle at its origin. In my opinion the failure of this operation to produce a complete paralysis is accounted for by the fascial bands which connect this muscle with the inferior rectus. The cut end becomes adherent at this point, thereby preserving some of the action of the muscle. This theory has been substantiated by surgical exploration failing to reveal any orbital attachment in many of the cases with persistent upshoot. Therefore, if, by receding the inferior oblique a known number of millimeters, we can avoid a complete paralysis, yet at the same time obliterate all overaction, we shall have made definite progress. My experience

with this operation is too limited to give a final appraisal, but my impression is that it has a genuine field of usefulness. I have done it only five times, and these cases have not been followed long enough to pass final judgment upon them. I can say that in no instance has it produced a complete paralysis, and all cases have been benefited. I have used it only in those cases in which the external rectus had to be operated upon at the same time. With this muscle reflected out of the way, recession of the inferior oblique is not difficult. Doing this operation on the cadaver with the external rectus in situ has convinced me that it is a much more complicated procedure than the myomectomy.

While discussing the inferior oblique, may I remind you that it is frequently adherent to the lower border of the external rectus as it passes underneath this muscle to reach its scleral insertion. The fusion of these two muscles at this point may lead to an unintentional and unrecognized severance of the oblique in the course of a resection of the external rectus. I feel that such an injury to the oblique does account for some of the vertical deviations discovered after a shortening of an external rectus.

Dr. Luther C. Peter, Philadelphia, Pa.: If and when an external rectus muscle is detached to correct a lateral deviation, the approach to the insertion of the inferior oblique is quite simple. As Dr. White has pointed out, under such circumstances it is logical to tenotomize the insertion of the oblique if this is indicated. To make this approach under other conditions is inadvisable. The usual approach through the skin is accompanied by little trauma and by slight risk of infection, as compared with operation on the insertion. It is true that tenotomy of the oblique at its origin does not always yield uniform results, but the same is true of any tenotomy, depending upon the complete severance of all fibers. The same variation is equally applicable, under like circumstances, to the insertion of the oblique.

Operation on the insertion of the inferior oblique, when operative interference is indicated, has, for obvious reasons, more to commend it than similar operations on the insertion of the superior oblique. In the first place, detachment of the superior rectus becomes necessary — a questionable procedure — in order to gain access to the superior oblique. Second, there is a definite anatomic connection between the superior rectus and the superior oblique, when these muscles cross. This led Dr. Wheeler to advise rather free surgery in order to gain the desired results, especially in shortening of the superior oblique.

Those who do much retinal separation surgery have realized the wisdom of Dr. White's proposal when the external rectus must be detached. Under the proper conditions it is a worthwhile addition to inferior oblique surgery. It again emphasizes the careful selection of the proper muscle operation to accomplish definite ends.

It is gratifying to me to learn, from Dr. White's closing remarks, that he now recedes the inferior oblique. It is quite evident to any of us who do much muscle surgery that tenotomy of the vertical acting recti is not indicated. It is really a serious thing to do, and the same is true of the oblique, unless one is dealing with an overacting oblique and you have a superior rectus palsy. If one expects to get a full paralysis of this muscle—and apparently Dr. White's experience has led him to the conclusion, as has also that of Dr. Dunnington — by tenotomizing the tendon at its insertion, this paralysis is more nearly complete. I believe, therefore, that is a definite indication for the operation which Dr. White has so well devised.

Dr. Conrad Berens, New York, N. Y.: I heard one of Dr. White's students speak of his suggested operation in 1936 or 1937. It was not until 1938 that I attempted this procedure, and since then I have performed unilateral recession of the inferior oblique of only one eye on eight patients; bilateral recession of the inferior oblique, in one case, and inferior oblique surgery combined with operation in two cases. This is a total of 11 operations. Because of the importance of the new procedure, I have had my results analyzed to discover just what may be obtained from the operation on patients without other rectus surgery, and I have done what Dr. Peter says we should not do, and found it not too difficult; that is, to operate on the inferior oblique without detaching the lateral rectus, but, by grasping the inferior oblique with a special muscle clamp, you can detach it at its insertion. When you detach it completely, you may have trouble because it

is difficult to see where the muscle was attached. A method which has been most satisfactory is to take a small tenotomy hook and lightly cauterize the sclera at the anterior extremity of the inferior oblique insertion. In all but two of these patients nylon sutures (8-0) were used without reaction. Experimental studies with nylon sutures have been in progress for about a year, and these sutures apparently produce as little irritation as any suture material now available to us.

So far as the results are concerned with tenotomies and myectomies of the inferior oblique that Dr. White has objected to, I have similar objections. One of these objections is that some of these cases have developed cyclotropia. So far, in the 11 patients I have operated upon by recession I have not observed serious cyclotropia. The greatest objection I have had thus far is that I have not placed the inferior oblique back far enough. I have not receded it more than 7 mm., the majority having been from 5 to 7 mm. Unless the lateral rectus is detached it is very hard to work in this particular field, and that is probably one of the reasons why I have found it difficult to recede the inferior oblique more than 7mm. When the lateral rectus is detached, it is easy to suture the muscle as far from the insertion as you wish.

Of the eight patients upon whom I performed unilateral recession of the inferior oblique, five showed marked improvement — from 4 to 6 prism-diopters to total correction of 18 to 20 prism-diopters, which brings out the point which Dr. White made: we still do not know how far to recede the muscle. None of these patients was amblyopic, and therefore the deviation could be measured carefully pre-operatively and post-operatively. One patient who had a bilateral hypertropia was corrected insufficiently. Two cases showed some increase of hypertropia, one of which was operated upon again. It was a somewhat small recession (3 mm.), and later I performed myectomy of the inferior oblique with marked improvement.

It is interesting, as Dr. White pointed out, and as Duane has stated, that there is a diminution of the lateral deviation with correction of the vertical imbalance. In these eight cases in which the inferior oblique alone was operated upon there was a reduction of the horizontal imbalance (esotropia) from one-third to one-half of the deviation as measured with the screen test and square prisms.

In addition to the unilateral operations on the inferior oblique, there was one case of bilateral recession of the inferior oblique — 3 mm. on one side and 3.5 mm. on the other. This patient had a left hypertropia of 25 prism-diopters and a right hypertropia of 9 prism-diopters, reduced respectively to 6 prism-diopters and 5 prism-diopters. The lateral imbalance also improved from an esotropia of 18 prism-diopters to 5 prism-diopters.

In two cases multiple operations were performed, including a recession of the inferior oblique. One of the cases showed improvement in the hypertropia, the other had a transfer from a right hypertropia to a left hypertropia of the same degree.

From the work we have done so far in this particular field, we believe we have had a tendency toward undercorrection in most of our cases by an average retroplacement of from 5 to 7 mm. The improvement of the lateral imbalance has been marked in most of our cases, and that is something we should consider in performing this operation. I believe Dr. White has made an outstanding contribution to the surgery of the inferior oblique.

Dr. James Watson White, closing: In my effort to keep my paper within the required limit of time I omitted many details which I should like to have mentioned. Before a group — in my office last week — I talked on this subject for an hour and a half, and then I felt I had not covered it as satisfactorily as I should have liked, so a paper of fifteen minutes' length does not allow one much time.

I feel the same as Dr. Dunnington about the effect when you do a myomectomy or tenotomy at the origin, but one should be careful that the paralysis of the superior rectus is almost complete and that the upshoot is marked enough to warrant it. I have no statistics, but I have done many, and the great majority of them in which I have judged I could do a myomectomy or tenotomy at the origin have been satisfactorily corrected, and comparatively few overcorrected, but these few that were overcorrected I would now like to have receded. The first operation I did at the insertion was on a patient (D. D.), and I have not had a nicer result, so the muscle cannot be very difficult to find. I did not operate upon the lateral rectus at the same time. The patient still has

some esophoria, but I went in, as I indicated on the lantern slide, at a downward and outward angle of about 20 degrees, keeping below the border of the external rectus. Then, when the muscle is engaged, it is taken on two hooks which are separated. Once in a while you will find it tied up with the external rectus. If so, it is dissected away and one hook is pushed back to the insertion. Well toward the insertion I use a double armed suture and the muscle is cut beyond this. It is then receded to the distance believed necessary to secure the desired effect. It is not difficult, and if you think it is, try it on the next case in which you are going to do an enucleation, or if you are going to do an operation in the lower outer quadrant of the globe, endeavor to engage the muscle with a hook when doing an external rectus resection or recession; it does the inferior oblique no harm to pick it up and drop it back without tenotomizing it, to see if you can find it in about the same place. We have had no trouble finding it.

I have had no infections. I do not see why we should get an infection in any muscle operation, except as a rare accident. I have been more or less in touch with Dr. Berens' work at the New York Eye and Ear Infirmary, because some of the men there have been doing this operation at my suggestion and getting very satisfactory results.

Dr. Frank Keil varied the procedure in one case. After exposing the muscle, it was drawn slightly to one side and the scleral suture taken at the desired distance along the line of the muscle before it had been displaced. The double armed suture was then passed through the muscle, well toward the insertion. The muscle was then severed just beyond the suture. When the suture was tied, the muscle was replaced to the site of the scleral sutures.

This is not a bad idea, and I have suggested it to those who were afraid to perform the operation. We have done many of them and had no trouble. The surgeons at the Post-Graduate Hospital are doing them all the time.

The Effect of Mydriatics Upon the Intra-Ocular Pressure in So-Called Primary Wide-Angle Glaucoma. Dr. Peter C. Kronfeld, Chicago, Ill., Dr. H. Isabelle McGarry, Chicago, Ill. (by invitation) and Dr. Homer E. Smith, Chicago, Ill. (by invitation).

As the counterpart to the narrow-angle glaucoma which, since the advent of gonioscopy, has become a well understood and sharply defined entity, the term "wide-angle glaucoma" is used to comprise the various forms of so-called primary glaucoma in which the intra-ocular pressure is abnormally high in the presence of a grossly normal, unobstructed chamber angle (except for occasional pigment infiltration of the trabeculum). In eyes which probably came under the category of wide-angle glaucoma Koellner (Arch. f. Augenh. 88:58, 1921) showed that the intra-ocular pressure was, as a rule, not demonstrably influenced by the administration of mydriatics.

The present study represents a repetition of Koellner's work with the special aim of correlating the effect of homatropine upon the intra-ocular pressure with the gonioscopic picture and other features apparently characteristic of wide-angle glaucoma. The results permit sharper definitions of those forms of so-called primary glaucoma which may be "provoked" by mydriatics.

Discussion

Dr. S. Judd Beach, Portland, Me.: Dr. Kronfeld mentioned the report by Holt and myself on dilation glaucoma at last year's meeting. His paper represents another step in running down the subversive elements in this disturbance. Besides the narrow chamber angle, usually indicated by shallowness of the chamber remarked by us, it will be recalled that endocrines are implicated, as shown by the overwhelming predominance of female victims. Some other factor is still unknown. Hans Barkan suggested the menstrual cycle. That something of this kind exists is indicated by the observation of Magitot on the erratic behavior of provocative tests. He said, in reporting a series of cases, that in the same case an agent that will precipitate an exacerbation on one afternoon may have no effect whatever the following morning. Dr. Kronfeld's observations are of especial interest in supporting the preference of Scandinavian observers for the drinking test rather than mydriatics in uncovering latent glaucoma.

Some Observations on the Results of Desensitization in Tuberculous Iritis. Dr. E. V. L. Brown, Chicago, Ill., Dr. Ernest E. Irons, Chicago, Ill. (by invitation) and Dr. S. L. Rosenthal, Chicago, Ill. (by invitation).

In iritis "without etiology" tuberculin sometimes causes local, general and focal (eye) reactions. Theoretically the uvea is sensitized. If sensitiveness could be reduced recurrences might cease.

We attempted desensitization by inhalation of fumes from heated suspensions of dead tubercle bacilli in salt solution. In two cases of recurrent iritis definite reduction of sensitiveness was achieved, as determined by periodic titrations with intracutaneous tuberculin, and freedom from recurrences (21 months).

Two cases and two years do not furnish sufficient data on which to base conclusions. Our observations on animals lend considerable support, but for the present we attempt to tell only what we have observed.

Experimental Studies in Ocular Tuberculosis. VII. Effect of Desensitization with Tuberculin in Experimental Ocular Tuberculosis. Dr. Alan C. Woods, Baltimore, Md., and Dr. Earl L. Burky, Baltimore, Md. (by invitation).

The results of an experiment dealing with the effect of desensitization with tuberculin in ocular tuberculosis are reported. Eighty immune-allergic rabbits were inoculated in the eye with virulent tubercle bacilli. Twenty-six days after inoculation, when the tuberculous lesions were well established, the rabbits were divided into two groups with equally severe eye lesions. One group was treated with tuberculin. The second group was untreated. After an initial focal reaction, the treated group showed a more rapid subsidence of the ocular lesions and a greater incidence of healing than did the control group. The later determination of the ocular and cutaneous sensitivity in the two groups indicated that selective desensitization of the eyes resulted from the intracutaneous administration of tuberculin.

Discussion

Dr. Allen Greenwoood, Boston, Mass.: I would like to say that during the past twenty-five years at least I have followed the method of desensitization in all tubercular eyes, and I shall continue to do so. I am positive that I have had results from the procedure.

Dr. E. E. Irons, closing: In view of the newness of this approach and our lack of knowledge of the therapeutic action of these fumes, we have proceded very carefully; perhaps later we will learn that we have been overcareful. However, until we know more of the possible effects we still feel that the dosage should be regulated carefully, as described in the paper. No doubt, further experience by others will throw additional light on this question.

With respect to the nature of the material contained in these fumes, we know that when the fumes are passed through a fritted glass filter, so that presumably no tubercle bacilli, or fragments thereof, can be carried through, they still have the same effect on guinea pigs that the unfiltered fumes did. The distillate from these fumes, concentrated 30 times, does not neutralize tuberculin.

Dr. Alan C. Woods, closing: I would like to say this in appreciation of Dr. Brown and Dr. Irons' work. We are very far from knowing the proper methods for the use of tuberculin for desensitization. Evidence is gradually accumulating that the intracutaneous method may be preferable to the subcutaneous method. There is a very peculiar class of cases which we are totally unable to desensitize. Even the briefest exposure to tuberculin produces violent reactions. There is a possibility that we may be able, with histaminase, to produce a negative phase in these patients and be able later to give them tuberculin.

While Dr. Brown's work requires a great deal of standardization before we will dare use it clinically, it may be the secret to the proper approach to obtain desensitization. Possibly the alveoli of the lungs are the proper portals of entry. As I have already said to Dr. Brown, while one observation does not prove a point, nevertheless it gives one considerable encouragement to know there are additional therapeutic possibilities.

Dr. E. V. L. Brown, closing: I wish to disclaim any credit for this work—it is all Dr. Irons'. This is iritis, and it is modified. I am only the the ophthalmologist; it is Dr. Irons' work.

Allergic Keratoconjunctivitis. Dr. C. S. O'Brien, Iowa City, Iowa, and Dr. J. H. Allen, Iowa City, Iowa (by invitation).

An unusual type of corneal lesion occurs as a result of contact with various allergens, e.g., butyn, pontocaine, atro-

pine, lanolin, sulfathiazole, oil of orange and others. This lesion appears as a subepithelial infiltrate in the peripheral areas of the cornea. As a rule there is an accompanying conjunctivitis and often inflammation and swelling of the lids. Diagnosis is difficult and the cause not easily determined. Removal of the offending agent results in rapid recovery.

Discussion

Dr. Francis Heed Adler, Philadelphia, Pa.: In the office I use pontocaine altogether for local anesthesia, and we have seen a number of cases of pontocaine sensitivity. Last year I had the unpleasant task of enucleating an eye which I am certain was lost due to sensitivity to pontocaine. This patient consulted another ophthalmologist for a cyst on his lid, and I believe pontocaine was used at that time. The patient stated that a local anesthetic was instilled and he developed a conjunctivitis. He returned to the ophthalmologist, who said he had pink eye; the conjunctivitis grew worse, and he went back again and was given a prescription for pontocaine to put in the eye. A lesion appeared on the lid, and a dermatologist was called in, who made a diagnosis of pompholyx. I believe that is an old-fashioned term for any vesicle on the skin — a contact dermatitis. The condition gradually grew worse; the patient developed a corneal ulcer, and was seen in consultation by a number of physicians in Philadelphia. He then went to New York and was seen by Dr. Knapp, who suspected that he was sensitive to pontocaine. By that time the eye was ready for enucleation. He went to Florida, where Dr. Black saw him. Finally he ended up with a chronically inflamed, sightless eye, which I removed. One day after the enucleation when he came in the office he had quite a bit of secretion in the socket, and I wanted to paint the lids with an astringent. I unwittingly instilled a drop of pontocaine in the socket. I washed it out immediately, and he knew nothing about it. He called me up in an hour saying that I had put something in his eye, and that he had the same reaction he had had before and would I see him right away. I did, and he had it alright; so we know that he is sensitive to pontocaine. I believe this is much more frequent than we know, and from now on I am on the alert for patients who are sensitive to pontocaine.

Dr .William Tarun, Baltimore, Md.: I have several colored slides, taken at different times, of a patient I saw in 1934 in consultation with one of the younger men in Baltimore. The patient, a physician, complained of having something in his left eye for which a solution of pontocaine four times a day had been prescribed. This was supplemented by merthiolate and atropine. Ten days after the onset of the symptoms I was asked to see him and found the subjective symptoms were severe pain, slight visual disturbance and marked lacrimation. The objective symptoms were swelling of the lids and numerous hemorrhages of the ocular and lid conjunctiva, with marked proliferation of the tissues on the upper tarsal plate, somewhat resembling an acute trachoma. The cornea showed a slight membrane at the temporal limbus, but the cornea itself had a fairly good luster. It appeared to me to be due to some chemical irritation — I must confess I did not suspect pontocaine. It occurred to me, however, that it might be due to merthiolate or emetin, the latter having been prescribed for the patient because of amebic dysentery a few days before the onset of his eye symptoms.

Since I had never used merthiolate or pontocaine, these were immediately discontinued, and holocain, one-half of one per cent, was substituted. Cold compresses and atropine were also suggested. Believing that the merthiolate and emetin were responsible, I instilled in a rabbit's eyes both of these drugs for a period of five days, but no change other than a slight conjunctival irritation resulted. The culture of the conjunctival sac was positive for pneumococcus, and a solution of ethyl hydrocuprein was prescribed, but this seemed to add more fuel to the fire and was discontinued.

In spite of treatment, the membrane continued to form in a concentric manner, as shown in the first slide. A section was removed, which, as reported by a pathologist of the University of Maryland, showed numerous epithelial cells. As the patient's condition became desperate, I asked Dr. Jonas Friedenwald to see him, and he expressed the opinion that it might be due to cocaine instillations, but these had not been used. He also suggested that the membrane be allowed to form completely over the cornea. In the second slide the cornea was completely covered with membrane except for a small central area. After about three weeks the eye be-

came quiescent, with the formation of a slightly hazy cornea and a reduction in the vision to 20/30. I had previously refracted this patient on a number of occasions. His refractions were those of a moderately high myopia with a visual improvement to 20/15 in each eye.

About three years later he returned complaining of a lump on his lower right lid. This proved to be a chalazion. In view of the fact that the cocaine usually dilates the pupils and causes a diminution in vision, I suggested the use of pontocaine solution. This was instilled several times. The lower lid was kept everted so it would not affect the entire cornea, and the cyst was opened and curetted. He was given a solution of sodium borate and instructed to use cold compresses. Later in the day I received a telephone call stating that he was suffering a great deal of pain, and, being of a somewhat nervous temperament, codeine was given for relief of pain. When he presented himself the following morning the lower lid was swollen, and on the lid and ocular conjunctiva there were numerous hemorrhages; the cornea showed a slight loss of luster, which stained readily by fluorescein, and he was given holocaine to use every hour. Within a few days he made a complete recovery with no loss of vision. I have no doubt that this was definitely a case of pontocaine sensitivity.

Dr. Wilmer, some years ago, reported before this Society a series of 500 cases in which pontocaine had been used, without a single case of sensitivity. I must say I was misled by his report, and did not suspect pontocaine at this time. In foreign literature a number of cases have been reported, but to my knowledge only one case in the United States. This report was found in the Archives of Ophthalmology following my case. Investigators, however, have proved one thing, which should be borne in mind, i.e., that local anesthetics bear a certain relation to the tissues: the more rapid the effect of the anesthetic, the greater is the destruction of tissue. I am very confident that nothing will take the place of cocaine and holocaine in ophthalmic operations.

Dr. F. H. Verhoeff, Boston, Mass.: My experience agrees with that of Dr. O'Brien, except that I believe I have seen worse cases than he has described. I recall particularly one case due to atropine. A good many years ago I had a patient who knew she was sensitive to atropine, but she did not tell us anything about it, and so, as a result of not more than two drops in one eye, she lost this eye. She had a tremendous dermatitis. Her face looked as though she had erysipelas; she had a keratitis which went on to complete ulceration of the cornea, and I proved that it was due to atropine. I asked the intern to test her, and he injected 1 per cent atropine into the skin of her arm. As there was no reaction in twenty-four hours, he then injected 2 per cent in the same arm. In another twenty-four hours the whole arm was a mass of inflammation. Since then I recall two cases of large ulcers of the cornea following the use of atropine. The patients came in with these ulcers, and were still using atropine. On discontinuing the atropine these ulcers cleared up promptly, proving that they were due to the atropine. In vernal catarrh we very rarely get corneal involvement, but the same type of ulcer does sometimes occur in this condition. These ulcers are characteristic, but right now I can't remember exactly what these characteristics are. I called Dr. Gunderson's attention to them, and he remembers what they are. I am sorry that he is not here to describe them. The ulcers may be large and may encroach on the pupillary area. The ulcers which Dr. O'Brien described are not serious, and they heal without causing loss of vision.

In connection with pontocaine, I may say that I had a patient on whom I used it for taking intra-ocular pressure, and she had a marked reaction after she went home. She came back and complained that it was the drops I had put in her eye that caused it. I used holocaine the next time, and she did not have any reaction. The next time I forgot and used pontocaine again, and she had another reaction. I believe that happened once more. Then I told her that if she ever got the reaction again it would be her own fault, because she should remind me not to use pontocaine.

Dr. John Green, St. Louis, Mo.: Some years ago I was consulted by a woman who had had conjunctivitis for a week or more. She had been in the charge of a colleague who had used ordinary measures without improvement. For the first few days my treatment was equally unsuccessful. One morning she volunteered the information that every time she wore a fur coat which she had purchased the

preceding summer she developed a facial eruption resembling acne. That suggested the possibility that the fur coat might have some bearing on the ocular condition. The patient was referred to a dermatologic colleague who said: "This case is similar to several I have seen recently; some women who wear iron-dyed fur coats develop a skin eruption, often associated with conjunctivitis." After that the cure was simple. On request of the dermatologist, the furrier exchanged the fur coat for another that did not contain this particular iron dye. The patient promptly recovered without local medication.

Last December, as a woman was smoking a cigarette, someone jarred her elbow and cigarette ashes dropped into her eye. A small central corneal denudation was found. The pain was not severe. The lesion healed in a few days without the use of local anesthetics. Three months later, while shopping, the pain returned. She appeared at the office with a central superficial corneal lesion. There was no foreign material. The pain that day and the following was very intense, and she was given 0.5 per cent pontocaine solution to use as frequently as necessary. On the third day I was startled to find an intense edema of the eyelids which extended to the cheek. A patch test with pontocaine was strongly positive. Holocaine, 1 per cent, replaced pontocaine, followed by prompt subsidence of all signs of the allergic reaction.

Dr. Alan C. Woods, Baltimore, Md.: Our experience with these corneal and conjunctival lesions has been quite identical with those described by Dr. O'Brien. When it comes to the detection of the specific allergin responsible, there are one or two points of some practical interest. Sensitivity to the inhalants, foods and bacterial toxins can be better detected by the scratch test. Drug sensitivities can be better detected by the patch test. The detection of a local conjunctival or ocular sensitivity can usually be done quite safely by the instillation in the eye of extremely weak dilutions of the suspected allergin, thus provoking a local reaction which helps identify the allergins responsible. As to pontocaine sensitivity, I believe the situation is not so black as it has been painted. While we have found butyn and atropine sensitivity to be fairly common, pontocaine sensitivity has, in our experience, been comparatively rare. In a great number of cases where pontocaine is used as a local anesthetic we have seen only three cases of sensitivity during the last three or four years. There are certainly several thous and cases in which it has been used routinely. The mechanism of this sensitivity has been clearly worked out. Landsteiner, working with guinea pigs, found that animals could not be sensitized to arsphenamine, but could be sensitized to a mixture of guinea-pig serum with arsphenamine. This applied also to various heavy metals and silk, the mixture apparently denaturing the native protein.

Apparently this is what occurs in the eye: These patients are not sensitive at first, but with the action of the tears on the alkaloid a foreign protein is formed to which the patients develop a sensitivity. That may be the reason, as Dr. O'Brien has pointed out, that in many cases the sensitivity is a local one and it is very difficult to detect, unless you use weak dilutions of the allergins directly in the conjunctival sac, increasing the strength until a local reaction is obtained. I believe that if this is done carefully, it is quite safe and it is sometimes the only method by which we can demonstrate a specific allergic response.

Dr. William H. Crisp, Denver, Colo.: In one or two surgical cases I had a distinct conjunctival and lid edema reaction to 1:5000 merthiolate ointment.

Dr. Eugene M. Blake, New Haven, Conn.: Will Dr. O'Brien say something about nupercaine, which I have used many times a day for at least ten years, and I do not think I have seen any such reaction.

Dr. Sanford R. Gifford, Chicago, Ill.: I would just make a suggestion which may help in the prevention of these induced cases, such as Dr. Verhoeff described. I have given butyn to patients who I knew were sensitive because I had forgotten they were sensitive, but we now have put on every sheet of such patients' records, in red type, "No butyn" or "No pontocaine." Even then we forget at times, and always tell the patient to remind us not to use butyn or pontocaine.

Dr. C. S. O'Brien, closing: I am sure the discussion has been more interesting than the paper. I knew that these cases were being seen, but I believe it is well

to bring them to our attention occasionally.

I am sorry to say we have not used nupercaine at all, Dr. Blake.

I am in complete agreement with Dr. Woods' discussion, but I have found that pontocaine has produced quite a few of these lid and conjunctival inflammations. As a consequence, I do not use pontocaine. I believe it is a good anesthetic, but I would rather not use it. That differs somewhat from the experience of others, perhaps. Also apropos of Dr. Woods' discussion, we used weak solutions of these drugs in the conjunctival sac, as stated in the paper.

Corneal Vascularization Problems. Dr. Derrick Vail, Cincinnati, Ohio, and Dr. K. W. Ascher, Cincinnati, Ohio (by invitation).

In the conjunctivo-scleral wedge a more or less extensive meshwork of vessels is normally present. Vascular arcades are given off which enter the transilluminable part of the limbus so that their final loops seem to be situated in corneal tissue which is, however, the conjunctivo-scleral wedge. Usually, the venous return takes place separately for each loop by its own efferent limb but quite often all or almost all blood returns by anastomosing veins which run concentrically with the limbus. For these vascular features the term "concentric collaterals" is proposed.

Concentric collaterals are engorged parts of the pre-existent limbal meshwork.

These vascular anastomoses occur at a percentage of 13.3 in a material consisting of persons with manifest, subclinical or suspected deficiency in different vitamins, observed in the Nutrition Clinic in Birmingham, Alabama. Their occurrence was found at a markedly higher percentage in the Cincinnati General Hospital Eye Clinic.

Concentric collaterals should not be mistaken for corneal vascularization.

Any kind of longstanding engorgement in conjunctival vessels may produce the formation of concentric collaterals. They probably are due to a hindrance of venous outflow and are probably a kind of collateral circulation leading to the conjunctival veins in the horizontal meridian because of overcrowding in the original venous system. The dilatation is probably connected with cellular changes and not alone due to stretching of the vessel wall. This fact, on the other hand, interferes with an early reduction of the collateral circulation, if once formed: the dilatation of these vessels persists after disappearance of the provoking cause.

All patients in one group showing concentric collaterals had signs of or gave a history of repeated conjunctivitis; none of them was free from signs of chronic conjunctivitis; some of them were addicted to alcohol. Pingueculae and hyalin deposits were frequently found in the bulbar conjunctivae of these patients even in younger people. Relationship between the most marked conjunctival changes and the location of the concentric collaterals seems to be present.

Discussion

Dr. Alexander E. MacDonald, Toronto, Canada: My introduction to riboflavin deficiency disease was in a physician from the Connaught Laboratory of Toronto. He had been in the southern states and had been seen by Dr. Sydenstricker. I had not seen the vessels in his cornea, possibly because I looked only at the central area with the slit-lamp. I have examined the limbus many times for slowing of the circulation in retinitis pigmentosa, and even now, after hundreds of examinations in this region, I have difficulty in locating the exact boundary of the limbus. We must recognize three different limbus regions; clinically we recognize the line of junction of the sclera and the cornea. Under the microscope, pathologically it is an area at least 1 mm. in extent, beyond the limit of Bowman's membrane, where the epithelium develops an entirely different form, with papillae. With the slit-lamp there is still another limbus that must be further defined, and this is where the difficulty occurs, in trying to make a diagnosis of ocular ariboflavinosis from the invasion of the cornea by blood vessels. When Dr. Sydenstricker was in Toronto two months ago, he saw both these patients, and drawings of their limbus regions were shown by lantern slide. In the drawings the artist showed the beam in the cornea and the level of the vessels. You will see that they are superficial, with fragmentation of the circulation in the vessel as it passes forward into the cornea. The venous return loop is deeper, as are the vessels tangent to the limbus. Both these patients have lost much vision in one eye.

Ariboflavinosis is a modern diagnosis, but riboflavin deficiencies are not at all modern, nor is corneal vascular invasion. Recently reviewed records of 100 pathologic eye examinations made in 1936 showed that vessels invaded the cornea in 37 cases, and it is not likely that all these were due to riboflavin deficiency. The disease is often of long duration and may be serious. Many of us have prescribed riboflavin for patients suffering from recurring lesions of the cornea, ulcers that stain over extended periods, typical skin eruptions, etc., and the improvement with riboflavin (5 mg. a day) is amazing. The only intravenous riboflavin available in Canada is combined with nicotinamide, 4 mg. of riboflavin and 200 mg. nicotinamide, and we have not used it frequently.

The second lantern slide showed another corneal vascular type which Dr. Sydenstricker believed was an advanced case of riboflavin deficiency. The patient had recurring ulcers due to being on a "relief" diet, which is good though deficient in riboflavin. I am not particularly satisfied with these drawings, but they will illustrate the difficulties encountered in considering for comparison this debatable region. However, it is necessary that different groups of investigators compare their findings on this important subject.

Dr. K. W. Ascher, Cincinnati, O.: I would like to ask Dr. MacDonald whether his first picture corresponds to a case with an entirely clear cornea, or whether there were some corneal opacities or scars.

Dr. MacDonald: There was a very faint nebula.

Dr. Ascher, closing: That is very interesting in proving that some condition has preceded the proliferation of the vessels. I think there is no difference in our opinions except that I want to stress the fact that there is never a real corneal vascularization without a preceding corneal disease.

I would like to add a slide showing the frequency of concentric collaterals in the patients observed in Birmingham and in Cincinnati. In Cincinnati, concentric collaterals occurred at a frequency of about one-half of all cases investigated, whereas in Birmingham they occurred at a frequency of only 14 per cent in material that came from the Nutrition Clinic of Dr. Spiess, who studies patients with different kinds of vitamin deficiencies. In Cincinnati the patients were asked about their nutritional habits and were placed into three groups of diets: very good, good and poor. In the group of very poor diets there were 11 patients with concentric collaterals and 17 without; in the group of food diets 14 positive cases and 10 negative were present. This distribution, in my opinion, contradicts the nutritional etiology of the concentric collaterals. Even the occurrence of concentric collaterals in the individual patient was not linked with nutrition; for instance, we had 10 bilateral cases in the group of good diets and only five in the group of poor diets; unilateral cases were four in the good diet group and six in the poor diet group.

This is only a preliminary study on 69 patients, selected at random in the Eye Clinic of Cincinnati; so it could happen that a later series would have another distribution; but the cases in Birmingham were more than 640, and they came from material of more or less severe deficiency diseases—some of them with manifest pellagra or with beriberi — and in spite of this fact the percentage of patients with concentric collaterals was only 14.

Local Sulfonamide Therapy of Catarrhal Conjunctivitis. Dr. Phillips Thygeson, New York, N. Y., and Dr. Alson E. Braley, New York, N. Y. (by invitation).

Experiments on the treatment of acute and chronic catarrhal conjunctivitis at Vanderbilt Clinic and Letchworth Village with sulfanilamide, sulfathiazole and sulfadiazine ointments are reported. Of these, sulfathiazole ointment in 5 per cent concentration proved most satisfactory and was found to be equal or superior to ordinary methods of therapy in conjunctivitis due to diplococcus pneumoniae, streptococcus hemolyticus, staphylococcus aureus, bacillus influenzae and Moraxella duplex (diplobacillus). Sulfathiazole ointment was particularly efficacious in zinc sulfate resistant chronic conjunctivitis in which both diplo-bacilli and pathogenic Staphylococci occurred simultaneously. A few allergic reactions to the sulfonamides and to the ointment bases were encountered.

Discussion

Dr. Sanford R. Gifford, Chicago, Ill.: I am sure we are all indebted to Dr. Thygeson for the stimulation which his excellent work on conjunctivitis has given to the exact diagnosis in chronic cases of this disease. I believe that this has been

a neglected field of ophthalmology for many years, until he has made us, with the use of the sulfonamide drugs, come to realize the importance of making an etiologic diagnosis. I have been following, as well as I could, a series of cases in which I have been using sulfathiazole locally. I have used it chiefly in cases in which I could make the diagnosis of staphylococcic conjunctivitis on culture with mannite-containing media in which there were strains of Staphylococci which fermented mannite. When cultures are difficult to obtain (some of you may not have facilities for making them in your offices, and they are most necessary in these cases), the patients in which one would make a tentative diagnosis of staphylococcic conjunctivitis are those cases associated with chronic blepharitis, recurring styes, and where these filtrates occur around the limbus. I have had such a condition myself, so I have been especially interested in it. I arrived at the International Congress in Cairo with the largest stye on record and was very much humiliated. The way I have treated these cases is something like that which Dr. Thygeson describes, except that I have found it inconvenient to use ointment during the daytime. Most of my patients are greatly inconvenienced by it, and I have used a 3 per cent solution of sodium sulfathiazole five or six times during the day, and a 5 per cent sulfathiazole cream at night, as put out by one manufacturer. A good many of these cases have responded well.

I have also experienced, as Dr. Thygeson has, cases of allergy to sulfathiazole in which just when we believe the patient is perfectly well he comes in with an attack which was apparently precipitated by the treatment. At least we have no way of knowing whether it was a recurrence or a result of the treatment, except by checking with cultures. If the culture is negative some time after the drug has been used, we can presume that this attack was the result of sensitization, and we should discontinue the sulfathiazole and use a mild alkaline wash, and see if the condition does not clear up.

The question is, how long should we use the sulfathiazole before that condition is likely to occur. I seldom use it longer than a month, and then change to a mild alkaline solution to keep the conjunctiva clean. In some cases I have carried out Dr. Thygeson's previous suggestion of using staphylococcus toxoid along with it.

Precancerous Melanosis and Malignant Melanoma of the Conjunctiva and Skin of the Lids. Dr. Algernon B. Reese, New York, N. Y.

A previous report on this subject was presented before this Society in 1937. The object of this report is to give additional evidence and experience to substantiate the original thesis that acquired conjunctival melanosis occurring in middle age is a precancerous lesion and the precursor of malignant melanoma. In the precancerous stage the lesion can be arrested by irradiation. It may also undergo spontaneous regression. The lesion arises from the basal layer of the conjunctiva. Naevi are different lesions clinically and histologically and practically never develop into malignant melanomas.

Discussion

Dr. Alexander E. MacDonald, Toronto, Canada: In our series of 100 malignant pigmented tumors at the Banting Institute we encountered one distinctly different in structure. There was a tendency to form gland-like structures, all the cells of which were heavily pigmented, so that the term melanotic carcinoma was justified. All other tumors in the series were of the sarcoma type.

The following is a report on the case of a printer, aged sixty years, who said that he noticed a dark spot at the edge of the lower lid and believed that it was printer's ink in his eye.

Before exenteration of the orbit the pigmented masses were present on both upper and lower lids, possibly by transplantation, as the canthus regions were clear of tumor cells.

The first lantern slide showed a vertical section of lids and globe **in situ**, with the heavy pigmented areas in both lids. The second slide showed a high-power magnification of the masses of pigmented cells arranged in gland-like form. Thus it seems that the conjunctiva may be the seat of melanotic carcinoma, and the use of this term is justified rather than the noncommittal designation "malignant melanoma."

Dr. S. Judd Beach, Portland, Me.: I should like to ask Dr. Reese a question. In one of these cases—and I am under the impression it was the one I saw with him—there was the history that recession took place after an attack of an acute infection. The recession which took place

was not only in the melanosis of the eye, but also in a pigmented mole on the body, and I would like to know if he has any comment to make on the significance of that history.

Dr. Theodore L. Terry, Boston, Mass.: In using an ultraviolet lamp with an ultraviolet filter one often finds that lesions containing melanin appear to be considerably larger, apparently resulting from fluorescence of "pre-melanin" in a true melanotic color. This is more striking with the mercury vapor lamp than with the carbon arc lamp.

Dr. Algernon B. Reese, closing: Dr. MacDonald's case seems to belong to the group discussed here.

Dr. Beach mentioned the spontaneous regression of a pigmented mole elsewhere in the body in the case which we saw together, following an acute infection. I do not remember the acute infectious incident, but the disappearance of a pigmented mole in the pre-auricular region occurred at the same time.

Dr. Terry's mention of his observation concerning the fluorescence of these lesions is very interesting.

Metastatic Orbital Infection: Report of a Case. Dr. Frank E. Burch, St. Paul, Minn.

In the case presented, a staphylococcic septicemia, emanating from a furuncle with metastatic foci in the chest, hip and ankle, produced a phlebitis of the orbit. A subacute exophthalmos, with alarming evidence of thrombophlebitis of orbital veins, without signs of infection in the accessory sinuses, led to exploration, drainage and ultimate recovery from the cellulitis. Sulfathiazole was of value in saving life as well as in controlling the local infection, but without operation could not relieve the abscess of the orbit.

Discussion

Dr. Alexander E. MacDonald, Toronto, Canada: There have been two important advances in the treatment of severe orbital infection, and complications formerly met are now rare. This preparation (lantern slide) dates back to 1926, and shows a large orbital abscess from a celloidin section of the orbital contents. The hematoxylin stain picks out the masses of pus cells to the temporal side of the optic nerve. The patient was a woman, aged thirty-two years, who had three teeth extracted from the lower right jaw. Three days later she was admitted to the hospital with a swollen right cheek. In three days her right globe became proptosed and fixed, and the lids swollen. The right cavernous sinus was drained, but the left eye became fixed and proptosed and death ensued in one week.

The staphylococcus aureus infection apparently was by direct extension through the pterygoid plexus, orbital veins and cavernous sinus to the left orbit.

The lantern slide section demonstrated the abscess of the orbit and the thrombosed cavernous sinus. A clouding of the posterior vitreous appeared when the globe became fixed, and the retinal veins were greatly enlarged. This picture of rapid extension of infection, it seems, can now be prevented by the use of heparin to stop thrombosis, and the use of the sulfa drugs to control the infection.

Dr. Eugene M. Blake, New Haven, Conn.: On March 21, 1942, I saw a case which was similar to the case reported by Dr. Burch. A boy, aged fifteen years, of Italian extraction, was sent to me because of increasing prominence of the left eye of two months' duration. The symptoms began with sudden diplopia while observing a motion picture. There was an exophthalmos of 4 mm. of the left eye, and decreased motility, especially in outward rotation. Because of severe headaches over the left eye and extending back to the occiput, several attacks of vomiting and the absence of fever or redness in the eye, a neoplasm was at first suspected.

Later the father of the patient stated that just before the onset of the exophthalmos the boy had had two boils on the back of the neck and he had squeezed these out. This was followed by a chill.

The patient was sent into the hospital for study and possible operation. Physical examination, except for the eye findings, was essentially negative. There was slight coryza, but no evidence of accessory sinus disease; teeth were good; tonsils were enlarged, submerged and diseased. Pupils were equal and reacted normally; fundi normal except for slight distention of veins of the left eye. X-rays revealed no evidence of tumor of the orbit or sinus disease. The white blood cell count was 16,300; lumbar puncture showed a pressure of 150 mm.

After entrance into the hospital there was a somewhat rapid increase in the exophthalmos, increase of pain, headache and redness of globe. The boy was prepared for a Kronlein operation, if necessary. However, an incision into the orbit between the globe and the lateral wall of the orbit was followed by evacuation of thick pus and subsidence of symptoms. Culture disclosed a hemolytic streptococcus albus and a non-hemolytic streptococcus. Sulfadiazine level was maintained at 9.7 mg. up to 10.4 mg. per cent before and after the operation.

The boy made an uneventful recovery, with disappearance of the exophthalmos and return of motility almost to normal at present, and vision of 20/20.

Observations on the Physiology of the Aqueous in Completely Iridectomized Eyes. Dr. Harold G. Scheie, Philadelphia, Pa., Dr. Frances H. Adler, Philadelphia, Pa., and Elinor Moore, Ph.D. (by invitation).

A method is described for the complete removal of the iris in cats. After recovering, the following conditions were found:

1. The eyes were free from any evidence of inflammation.
2. The intra-ocular pressure was consistently low.
3. The volume of the anterior chamber was reduced to approximately one-half that of the normal.
4. The protein content of the primary aqueous in the operated eye was equal to or slightly lower than that of the unoperated eye. In no case was it found to be increased.
5. The protein of the secondary aqueous was about 90 per cent lower in the operated eye than that of the secondary aqueous of the normal control eye.
6. The urea content of the operated eye was the same as that of the normal eye, i.e., consistenly lower than the blood level.
7. Blood was absorbed very slowly from the anterior chamber of eyes in which the iris had been totally removed.

These results are discussed in the light of our present knowledge of the physiology of the aqueous.

Discussion

Dr. Peter C. Kronfeld, Chicago, Ill.: If one withdraws aqueous from the human eye, there is a very definite limit to the amount that can be withdrawn. Toward the end of the aspiraiton the iris comes in contact with the cornea, or the iris is sucked into the tip of the needle. That can be avoided if the opening of the needle is made to face the cornea, yet even with the needle remaining unblocked one reaches a point where no more fluid can be obtained by moderate amounts of suction. In other words, there is such a thing as the aspirable amount of fluid. In certain eyes which are definitely diseased that is not the case, and the amount of fluid which can be aspirated is almost unlimited, vitreous fluid rapidly replacing the aqueous. In a recent paper by Irvine, Jr., of Los Angeles, that phenomenon was described in detail. The fact that in the eyes on which Dr. Adler and associates performed complete iridectomies, the amount of aspirable aqueous was definitely limited, I believe is an argument in favor of the normalcy of those eyes, in favor of the intactness, anatomically as well as functionally, of at least a large portion of those eyes.

In a very short time Dr. Adler showed us a number of figures which I was not able to digest, but I would like to ask him whether he cannot give us any figures on the rate at which the intra-ocular fluid was reformed after the aspiration of the primary aqueous in those irisless eyes.

Dr. Francis Heed Adler, closing: As far as we can tell, Dr. Kronfeld, the rate of reformation was approximately the same in the iridectomized eyes as in the normal eyes. However, that is, of course, more difficult to tell in an eye in which the iris has been removed, because it is more difficult to note the beginning reformation of the chamber unless you have a background like the iris from which to judge the depth of the chamber.

In slide 3 we had some figures showing the increased protein content in the plasmoid aqueous at different times, ranging from twenty-five minutes to twenty-five hours, so certainly within one-half an hour we were able to obtain sufficient fluid to begin our analyses.

A Colored Reflex from the Anterior Capsule of the Lens Which Occurs in Mercurialism. Dr. Walter S. Atkinson, Watertown, N. Y.

This reflex from the anterior capsule is lusterless and rather homogeneous looking and can be seen with the aid of the slit lamp. Its color varies from a brown-

ish gray to a deep rose brown and it is thought to be a deposit of mercury.

It was observed in all those examined who had symptoms of chronic mercurialism.

It is suggested as a diagnostic sign of mercurialism.

Discussion

Dr. Sanford R. Gifford, Chicago, Ill.: The case I am about to describe is just a freak case, which will not throw any light on this interesting subject. I saw this patient with my assistant, Dr. Droegemueller. The man was walking under a neon light, which broke, and part of it struck his glasses, causing a slight cut in his cornea, and the mercury from the neon light actually got into his anterior chamber. Three little globules could be seen there. The wound was small and closed immediately. It produced a very severe form of inflammation which was intractable to treatment. Unfortunately, the mercury was not removed at once, and later it was impossible to remove it. I have lost track of the case, but I believe the eye became practically blind and had to be removed because of these little globules of mercury which looked very innocent in the early stages. The inflammation became severe enough so that we could not detect these changes in the lens capsule which Dr. Atkinson described.

I had another freak case of mercurialism in a man who was dropping mercurochrome (2 per cent) in his eye, when he became very much salivated, and we did not know what was the matter with him, until we realized he was instilling mercurochrome 10 times a day and was actually absorbing enough to cause salivation.

Dr. R. von der Heydt, Chicago, Ill.: This is a very interesting field. The nearest analogous condition is chalcosis. In this we have a similar deposition. I think the term "deposition" is more nearly correct than reflex. In chalcosis we have a fine deposition of copper under the anterior lens capsule. This I was able to photograph (Am. Jour. Ophth., November, 1935). I had the opportunity of again seeing this case after six years. All the copper had been absorbed. The incrustation of the vitreous with copper, the involvement of Descemet's area at the limbus, as well as the subcapsular copper-colored disk, had disappeared.

I would like to ask Dr. Atkinson what happened in his cases: whether the eyes still show the so-called reflex after so many years, and I wonder why the reflex, or the mercury, was not carried away. The narrow slit-lamp beam might disclose where this deposition is. With copper the deposition is under the capsule, and it is not carried inward by the apposition of new lens fibers, so it must be in the lens epithelium.

Dr. Gifford has shown an eye with mercury in it, and I would ask if he has seen anything like this reflex in the lens. The eye had some metallic mercury carried into it by an explosion, and it remained there for a long time.

I want to thank Dr. Atkinson for this demonstration of ocular mercurialism.

Dr. John H. Dunnington, New York, N. Y.: Dr. Atkinson is to be congratulated on the discovery of this reflex in cases of mercurialism. It only goes to show that those who observe, see. I am sure it will prove to be a most important sign, for its frequent presence prior to the onset of other symptoms makes it of real diagnostic value. It was my good fortune to examine one of his patients and the picture is a most striking one. The dull gray appearance of the lens by oblique illumination would certainly lead one to expect a reduction in visual acuity. Then, with the corneal microscope, to see the varying colors in different areas of the anterior capsule is most striking. To my mind the most outstanding feature was the marked increase in density of the reflex in the pupillary area. With the knowledge that such a deposit occurs in mercurialism may we not expect similar reflexes to be found in chronic poisoning from some of the other heavy metals? However, so far as I have been able to learn, no such descriptions have appeared in the literature.

I would like Dr. Atkinson to tell us something of the experimental work he has been doing in an effort to produce this same type of reflex in animals exposed to mercury vapor, and I thank him again for bringing this to our attention.

Dr. K. W. Ascher, Cincinnati, Ohio: This most interesting paper raises some questions about the chemistry and physics of these deposits. So far as physics is concerned, there is an analogy in a kind of spectacles which have been recommended to reduce glare. They are not polaroid

glasses, but they are constructed by depositing finest metallic particles over the glasses. When you look at a person wearing a glass with these fine metallic deposits, you don't see the eyes of this person. The person looking through the glasses, however, is able to see very well and is not bothered by glare. This applies to the discrepancy mentioned by Dr. Atkinson, and concerning the objectively visible opacity of the lens as compared to the good visual acuity in these eyes.

So far as chemistry is concerned, there is an interesting question raised by Dr. von der Heydt, namely, why in Dr. Atkinson's cases the appearance of the mercury deposits did not change during the observation over many years. Copper deposits finally may be removed due to the solubility of the deposited copper salt. The mercury deposits are probably—I do not have any proof of this—insoluble compounds with lens proteins, and that may be the reason why they remain for a long time without being removed. We may even expect that later on, provided no further intoxication takes place, clear lens fibers will cover these strata of deposits.

There is another analogy to the mercury deposits so far as the localization and the aspect are concerned. It is a very rare one, and, so far as I remember, there is only one case of this kind that has been described by Ollof and Siemerling, about twenty years ago. They reported a case of Wilson's disease, where, besides the well-known deposits in the periphery of the cornea, there were deposits of similar color in the anterior lens capsule. Similar deposits were found by Fleischer in the lamina vitrea of the choroid. These observations show that the elastic membranes of the eye may be a point of predilection for the deposition of most different substances.

Dr. H. M. Burian, Hanover, N. H.: I should like to ask Dr. Atkinson whether he has any explanation why the periphery of the lens remains clear, or why the deposits are more dense in the center.

Dr. Walter S. Atkinson, closing: I am very grateful for the interesting discussion of my paper which has stimulated new thoughts regarding the problem.

Dr. von der Heydt asked regarding the duration of the reflex. Some patients were examined who had not worked with mercury for ten to twenty years, and the reflex was still present and just as vivid.

I had intended to include in the paper some matter regarding attempts to produce the reflex in animals, but the results were rather disappointing and proved nothing, so I did not include them. An attempt has been made to produce the colored reflex in animals, using rabbits and guinea pigs. The animals were placed in a cabinet each day for eight hours, but it was difficult to regulate the mercury content of the air at a uniform temperature and still maintain proper ventilation. Some of the animals died of what appeared to be acute mercury poisoning and asphyxia, and others of unknown cause.

More recently the mercury has been heated outside of the chamber in a closed vessel and the vapor carried to the chamber through a pipe. A temperature of 25° to 30° C. has been maintained in the chamber with adequate ventilation.

The concentration of mercury vapor is determined by an instrument designed by Swisher (Research Laboratory, General Electric Company) on the principle of the Nordlander instrument. With this instrument a concentration of one part of mercury vapor to one hundred million parts of air may be determined with an eight-hour exposure.

One animal (rabbit) has now survived four hundred and seventy-one hours in the chamber with the concentration of mercury, averaging approximately 2.00 mg. of mercury per cubic meter of air. In this animal there is a beginning faint gray reflex visible. When this becomes more pronounced, it is hoped by microscopic and chemical examination of the lenses to learn more about the exact location and form in which the mercury is deposited in the lenses.

Dr. Burian has asked why it is centrally located. I do not know. It appears that the iris seems to protect part of the lens, but just why more is deposited there is very difficult to explain.

Again I want to thank the members who have discussed this paper so interestingly.

Fibroblastic Overgrowth of Persistent Tunica Vasculosa Lentis in Infants Born Prematurely. II. Report of Cases — Clinical Aspects. Dr. T. L. Terry, Boston, Mass.

This disease differs from the unilateral condition in full term infants usually discovered a few days after birth. Four cases are reported in detail of infants all affected bilaterally, all quite premature, weigh-

ing less than 2,000 grams at birth, and all at least four months of age before the condition was noted. One similar case is included of an infant with the membrane in one eye and a lesion resembling chorioretinitis in the other.

Treatment includes high vitamin diet, x-ray, and operation to close the hyaloid artery by diathermy. Glaucoma has developed in two cases and separation of the retina has been observed in another. Results are discussed and a summary is given of other suggested therapeutic measures.

Discussion

Dr. Ralph I. Lloyd, Brooklyn, N. Y.: This is the first bilateral case of this type to come to my attention. A unilateral case of this kind has been under my observation for about fourteen years. This patient was four and one-half months old when first seen, and the diagnosis of pseudo-glioma was made because the opacity was at the posterior surface of the lens; its color was cold gray (not yellow white); there was a vessel carrying blood to be seen, and there was some pupillary response. Although a diagnosis of glioma had been made and immediate enucleation advised, the patient has been under observation since. This eye is evidently smaller than its fellow, the side of the face and the bony orbit is smaller, and the eye is farther back. The argument for retaining the eye of a child under these conditions is that further development of the orbit will occur. In any case where there is the slightest doubt as to malignancy and the eye is without vision, this argument has no weight. If this particular eye had been sacrificed in infancy (if the diagnosis had been doubtful), there would have been no loss, and only the inconvenience of an artificial eye. The micro-slides of another case show the mass of tissue on the posterior surface of the lens, and a large persistent hyaloid artery attached at the usual place. The second set of slides are from a case of Dr. Whalman, of Los Angeles. In addition to the mass on the back of the lens and the persistent hyaloid artery, the retina is totally detached and incloses the hyaloid stalk like a shroud. The cause for the detachment in these small children is not known, but it is a recognized type of pseudoglioma. Some years ago, at the Royal London Ophthalmic Hospital, 24 eyes were removed with the diagnosis of glioma, and of the group, seven were developmental defects of the type discussed here today.

Dr. Parker Heath, Detroit, Mich.: The approach to this problem by Dr. Terry is highly commendable. He discusses here largely the clinical phases of the problem, but he has also conducted a series of studies along experimental and embryologic lines, and has made a search of the literature. To me it is an outstanding study and contribution. None of us see enough of these cases to be very sure of ourselves clinically, and I believe all of us should contribute what we have of our own material to the studies of Dr. Terry, so that he may have a large pool for study.

On the screen is shown a section of an eye which came to our laboratory quite recently. The case was of the monocular variety, except that the other eye had a curious anomalous congenital arrangement of vessels on the nerve head and persistent glial tissue — the so-called Bergmeister's papilla—apparently a rather large mass of tissue partly on the nerve head. The specimen is poorly fixed, unfortunately; the retina is obviously detached, and this persistent hyaloid artery, covered with grayish material, is seen at the temporal border. According to the physician who sent the specimen in, the mass was continually enlarging, so he removed the eye, and justifiably so. This was a full-term infant. The lens is partly dropped out, but you can see clearly the retina and the mesenchymal membrane, and the next slide shows the pupillary end of it with the hyaloid artery and the detachment of the retina. As in Dr. Howard's case, these prolongations of fine fibrillae extend into the vitreous.

The next slide shows some hemorrhage and pigment migration and ingrowing vessels; it is a progressive affair. The retina showed the pseudo-rosettes, similar to the case reported by Verhoeff in 1906, wherein there were these pseudo-rosettes containing fibrillary structures in a malignant tumor of the ciliary body. This seems a transitional type of case, between bilateral involvement and the purely monocular involvement, and I feel quite sure, as evidence accumulates, that we will find more and more proof of inflammatory changes in these mesenchymal tissues, and, indeed, Dr. Terry pointed it out when he spoke of the development of synechiae. There are chronic in-

flammatory cells in some of these membranes.

The cases that have done relatively well—the few I have seen—have been those with the lens partly absorbed and disk-like, and some vision has been secured through a window cut in this membrane by means of scissors, not with a knife. The radial extensions back through the vitreous are so potentially dangerous that any tugging on this mass would detach the retina, if it had not detached before.

So we are confronted with the dilemma of either performing early operation or of leaving these cases alone, and with the evidence in favor of early treatment at present less than the benefits gained by delay. The eyes are small; the retina is undifferentiated usually. Leaving such eyes alone may show the retina detached —a potential glaucoma—and they have very little vision. There is no benefit from early operation from the standpoint of amblyopia, because the best vision one gets is below the level of the amblyopic eye, so that argument can be thrown in the discard.

Dr. R. von der Heydt, Chicago, Ill.: This contribution to ocular pathology is the most important of recent years.

Dr. Terry has brought out the fact that the tunica vasculosa lentis, which is destined to absorption, may result in an overgrowth in premature infants. Usually premature infants in hospitals are watched to determine whether they will live, not as to whether they have any abnormal ocular condition. I congratulate Dr. Terry on having discovered this condition in these very young infants.

I like the term "Mittendorf dots." Credit is thus given to Mittendorf, who first described these symmetric black dots on the back of the lens, although he did not know why they were there.

Dr. Terry's discussion was also very interesting, as to how the hyaloid artery normally stretches and bursts. We have a beautfiul slit-lamp picture of that in nature's physiologic disposition of the hyaloid artery remnant. The stretching gives it the spiral shape.

In deference to the memory of the late Dr. Francis Lane, who was the predecessor of Dr. Georgiana Dvorak-Theobald and the successor to Dr. E. V. L. Brown, I want to mention that his membership thesis to this Society was a study on "Persistent Fibrovascular Sheath."

There is one point I wish to re-emphasize which Dr. Terry brought out and which is important in differential diagnosis between his cases and glioma. A gliomatous mass may reach the back of the lens and can be seen as a vascular membrane; he drew attention to the fact that in persistent fibrovascular sheath the blood vessels will radiate from the center. This does not occur in retinoblastoma.

Dr. Sanford R. Gifford, Chicago, Ill.: I had a case like the one Dr. Terry showed, with a little round mass on the back of the lens (exactly similar to a case Dr. Leach had in Chicago). I enucleated the eye for probable retinoblastoma, and when we found out what it was, I looked back at my record and found the notation that the cornea had measured 9 mm. on that side, as compared to 11 mm. on the other side. Since then I have used that as a criterion, which I believe is of value in differentiating between this condition and glioma. I think it is true in nearly all gliomas that the cornea is of normal size. I would like to ask Dr. Terry if this is so. At least they don't have this degree of microphthalmos, and I believe all these cases will have some degree of microphthalmos. Even if you are going to take the eye out, which Dr. Lloyd suggests, and which I do not agree with, I believe we should endeavor to make a diagnosis. I have seen four other cases in which we have avoided taking the eye out and they are going along so far and have retained a fairly sightly eye, at least better than the result of enucleation.

Dr. Robert H. Courtney, Richmond, Va.: Several years ago a young woman who had been in an automobile accident had a complete evulsion of the optic nerve. After the accident, there was no bleeding or disturbance of the circulation. There was, however, a complete cone-shaped detachment of the retina, extending from the ora serrata to the macula and the disk. This shows that there can be a tremendous amount of stretching of the retina and of the vessels of the retina without any bleeding. This patient has been under my observation for a number of years, and there has been no evidence of deterioration of the retina during this time.

Abscess of the Crystalline Lens. Dr. Ralph O. Rychener, Memphis, Tenn., and Dr. Edward C. Ellett, Memphis, Tenn.

Purulent infiltration of the lens often accompanies perforating injuries of the globe, the diagnosis usually being made by pathological section following enucleation. The literature is briefly reviewed and a case reported in which there was primary abscess of the lens following intraocular foreign body. The report was illustrated by serial Kodachrome photographs. Chemotherapy and paracentesis caused resolution of the purulent infiltration. The eye became quiet with some retention of vision.

Discussion

Dr. Francis Heed Adler, Philadelphia, Pa.: The ordinary crystalline preparation of sulfa drugs cannot be injected with a hypodermic needle as the crystals are too large. Dr. B. F. Souders, in my department, conceived the idea that these crystals might be broken up by sonic vibration, and one of the research chemists in the Johnson Foundation succeeded in doing this and in producing a microcrystalline form of the drug. The importance of this was immediately recognized by the whole surgical division of the hospital, and the Smith Kline French Laboratories made a grant to the hospital for the study of these preparations. A report on the crystalline form of the drugs has already been made in the Journal of the American Medical Association in a paper by Chambers, Harris, Schumann and Ferguson. We have used the microcrystalline form in experimental intra-ocular injections and believe that it is of value. Some of the histologic sections, however, show changes in the retina, which we believe may be due to the drug itself, and accordingly we are not advising the intraocular injection of the microcrystalline form at the present time. In desperate cases, such as those just reported, this form of the drug might be of some use.

At present the chief usefulness of the microcrystalline form in ophthalmology seems to be the employment of the preparation in a lacrimal syringe for getting sulfa compounds into the lacrimal sac.

Dr. William Crisp, Denver, Colo.: I am not clear as to the use of the crystals. Are the crystals in solution?

Dr. Adler: In suspension. The crystals are small enough to go through the needle.

Cataract Complicating Corneal Scars After Perforating Ulcers. Dr. Bernard Samuels, New York, N. Y.

In a previous paper the writer discussed the changes produced in lenses in the active stage of purulent corneal ulcers. The present paper is a continuation of the subject. It describes the proliferative and degenerative changes that went on in the lenses of 36 globes after the healing of the corneal ulcers. The existence of the corneal scars cover a period ranging from a few months to 50 years. Many informative clinical and pathologic details are brought out.

Discussion

Dr. C. A. Clapp, Baltimore, Md.: I would like to ask Dr. Samuels if there is any scientific proof for what I understood him to say: The central subcapsular cells are nutritional and the peripheral ones are germinal; and if there is such scientific proof, just where the differentiation between these two portions exists.

Dr. Bernard Samuels, closing: In reply to Dr. Clapp's question, this paper deals with cataract from an anatomic rather than from a chemical standpoint. So far as metaplasia and abnormal products of the epithelial cells of the lens are concerned, as an exception an anterior subcapsular epithelial cell may produce a vesicular type of cell, such as is the characteristic abnormal product of a germinal cell at the equator. On the other hand, a germinal cell at the equator may produce a spindle-shaped type of cell, such as is the characteristic abnormal product of an anterior polar cataract.

Studies of the Fields of Vision in Cases of Rathke Pouch Tumors. Dr. Henry P. Wagener, Rochester, Minnesota, and Dr. J. Grafton Love, Rochester, Minnesota (by invitation).

For the present study a group of cases was selected in which successful surgical removal of tumors arising in Rathke's pouch had been carried out. In all cases records were available of the ophthalmoscopically visible lesions in the optic nerves and of the defects present in the fields of vision prior to and shortly after operation. In a few cases, the results of re-examination at a later date were available also. An analysis is attempted of the prognosis for recovery of vision in these cases.

Discussion

Dr. Walter I. Lillie, Philadelphia, Pa.: The cases reported by Dr. Wagener emphasized strikingly that ophthalmologists are too timid in making an early diagnosis of prechiasmal and chiasmal lesions from the visual field defects. The visual changes occur early, and in 94 per cent of the cases the characteristic visual field changes are present in one or both eyes. There are but few exceptions to this. The fundus changes may vary from normal to pallor of one or both disks, simple optic atrophy or choked disk. Choked disks are rare in mid-fossa lesions, and the absence of this fundus change should not rule out a space-taking lesion. Other examinations should be instituted early, including a complete roentgenologic and neurologic study. A Rathke pouch tumor has its anlage from rests of cells in the craniopharyngeal canal, and occurs usually in young individuals. A diminution of vision in one or both eyes is an early sign, and the patient usually first consults an ophthalmologist. When proper glasses do not improve central visual acuity to normal, careful perimetric field studies should be done. Only by this method can the early field changes, so characteristic of prechiasmal and chiasmal lesions, be elicited. When found, a complete general and neurologic examination should be done, and the patient referred as soon as possible to the neurosurgeon. The mortality rate is not high, and surgery is the only means of effecting a relief or cure. In these cases, if untreated, blindness follows somewhat rapidly, and if the patients survive, they become a public liability, and from an economic standpoint in states with Blind Pension laws greater tax burdens will result.

Dr. Allen Greenwood, Boston, Mass.: I have seen a number of these cases with Dr. Harvey Cushing during his later years in Boston, and I have been struck with the same thing which Dr. Lillie has been stressing.

Dr. Henry P. Wagener, closing: I wish to thank the discussers for their comments and to stress the point that, as Dr. Lillie and Dr. Greenwood stated, the fields of vision are of primary importance in the diagnosis. It is not wise or necessary to delay surgical intervention in all cases until the development of positive roentgenographic changes in the skull. Although it is commonly assumed that suprasellar calcification will be present in 85 to 90 per cent of craniopharyngiomas, yet in seven of the present group of 29 cases the roentgenograms of the head were normal at the time of operation.

Tumor Formation at the Disk. Report of a Case. Dr. W. E. Fry, Philadelphia, Pa., and Dr. P. De Long, Philadelphia, Pa.

This is the report of a tumor of the papilla with both an ophthalmoscopic and a pathologic report. The margins of the disk were raised about one and one-half diopters in the lower nasal quadrant. All margins were hazy and all appeared raised except the upper temporal quadrant. The retina became completely detached. Section of the eye disclosed a tumor mass within the papilla, separate from the choroid. The nuclei in this mass were enlarged and oval, with moderate variation in size. A small amount of pigment was present in some but not all sections.

Discussion

Dr. Bernard Samuels, New York, N. Y.: This case reminds me of one that I saw a few years ago at the New York Eye and Ear Infirmary. One eye showed a whitish mass over the optic nerve. It was not certain whether it was p a p i l l e d e m a. Inasmuch as it appeared to be solid and had a globular outline, it suggested a conglomerate tubercle of the optic nerve. The eye was enucleated, and the entire mass was found to be made up of epithelial cells that did not seem to be characteristic for any gland in the body. A few months later the patient was placed in an insane anylum. Shortly afterward he died. A postmortem examination revealed that the primary tumor was a carcinoma of the stomach.

Another case I have in mind was referred by a laryngologist. The patient had had pansinusitis for which a radical operation had been performed. In the choroid of one eye there were several whitish spots, slightly elevated and sharply defined. This patient was on his way to Europe. Soon after his arrival there he consulted Professor Siegrist in Berne. By that time the second eye was involved. A diagnosis of bilateral miliary tuberculosis was made. He was placed in a hospital, where in a short time he died of what was diagnosed as pneumonia. An autopsy revealed that the primary tumor from which

the eye lesions were derived was located in the lungs.

These two cases demonstrate that the first clinical manifestation of a tumor elsewhere in the body may be found in the fundus of an eye.

Dr. F. H. Verhoeff, Boston, Mass.: I saw the sections of this tumor, some which were not thrown on the screen here, and the latter are the more important. This tumor is regarded by the authors as a malignant melanoma, but I am sure that this diagnosis is incorrect. Sections show areas of neuroglia proliferated from the retina which had been folded up or injured in some way. The part of the growth that could have been the original tumor was not shown in the photographs. That part consisted of proliferating vessels and endothelium arising from them. Some of the pigment in the tumor seemed to arise from the retinal pigment epithelium, and the remainder was probably blood pigment. The nature of the pigment could have been determined by certain methods, but these were not applied, so that we cannot be sure just what the pigment was, but I am sure it was not pigment such as occurs in malignant melanoma. I think it is unfortunate that this tumor should get into the literature as a malignant melanoma of the papilla. In the first place, it is not a tumor of the papilla, but arose at the side of the papilla, and in the second place, it is not a melanoma.

Dr. Wilfred E. Fry, closing: I wish to thank Dr. Samuels for his discussion.

At the time Dr. Verhoeff was kind enough to look at the sections there were certain things that we tried to bring out which I do not think I brought out as well as I should. So far as the position of the tumor in the papilla is concerned, it does not seem to me that is very important. Certainly the papillary region is definitely involved. So far as the exact nature of the tumor is concerned, I am aware differences of opinion can exist there. I had the specimen examined by several pathologists in Philadelphia, who considered the tumor a malignant one, and at least one of them regarded the pigmentation in the tumor as important in arriving at a diagnosis. That was why we suggested that the tumor is a malignant melanoma.

Extrapapillary Coloboma. Dr. Ralph I. Lloyd, Brooklyn, N. Y.

This congenital and even familial defect may be mistaken for tuberculous choroiditis, hereditary macular degeneration or idiopathic choroiditis. These lesions are also the basis of claims for blindness of traumatic origin and recent losses of vision by policy holders of life insurance with disability clause attached.

Discussion

Dr. R. von der Heydt, Chicago, Ill.: There is one condition I would like to draw Dr. Lloyd's attention to that might be included in the differential diagnosis as a vague possibility, and that is a healed glioma. That sounds as though it may never occur. I refer you to an article by Axenfeld, who depicts such a case and diagnosed it so in Hirschberg's Zentralblatt (1916).

Dr. T. L. Terry, Boston, Mass.: As usual, Dr. Lloyd has again brought us a a subject of considerable interest. The point he brings out, that unscrupulous persons have used the existence of congenital visual defects to obtain undeserved settlements from insurance companies, is increasingly important, as the settlement of industrial accidents is becoming so considerable an economic factor. An example of this general type came to my attention in one patient, a former chauffeur, who was pensioned, for a time at least, by his former employer, when congenital color blindness was discovered in the patient. Elsewhere this color blindness was considered to have been acquired. When his pension was stopped, this man consulted me requesting that I furnish him with a statement to his former employer that his eyes were again normal in order that he could obtain from her a letter of recommendation enabling him to return to a former position.

Dr. Lloyd's illustrations are interesting and instructive. His discussion and terminology again bring up for consideration the possibility that the eye may rotate during development in order that the fetal fissure which is classically located downwardly and inwardly turns somewhat so that in the new position it is represented by the nerve fiber raphe extending temporally from the macula through the fovea, or that accessory coolbomas can exist extending toward the macula, as illustrated in the older textbooks.

PRESENTATION OF NEW INSTRUMENTS

A Confirmaiton Test for Refraction

Dr. Conrad Berens, New York, N. Y.: The confirmation test for the refractionist consists of two spheres or two cylinders, one plus and the other minus, mounted on one handle, so arranged that the examiner may check the patient's acceptance with great facility. This arrangement of lenses not only furnishes a means of rapidly checking the patient's answers, but it is also almost an essential in the trial case unless the ophthalmologist has more than one set of trial lenses. The pairs of lenses which have been found to be most universally useful are the plus and minus 0.25 D. spheres and cylinders, but spheres and cylinders are available from 0.25 D. to 1.00 D.

The handle is round and knurled, 62 mm. in length, and branched at one end to support lens rings for holding 38 mm. lenses. The handle is marked to show the strength of the lenses in diopters, and the lenses are also marked, the minus in red and the plus in white. In using these tests the plus and minus 0.25 cylinders are held in the right hand and the spheres in the left hand, and the accuracy of the correction is checked rapidly by placing the lenses before those in the trial frame and presenting first the plus and then the minus lenses merely by twirling the handle of the instrument. At times it is advantageous to superimpose the cylinders and spheres.

CANDIDATES' THESES

Progressive Myopia: A Possible Etiologic Factor. Dr. Edwin W. Burton, Charlottesville, Virginia

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Endophthalmitis with Secondary Glaucoma Accompanying Absorption of the Crystalline Lens. Dr. Robert H. Courtney, Richmond, Virginia

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Further Observations on Autofundoscopy (Auto-Ophthalmoscopy of Eber: Purkinje Figure of Walker). Dr. Leslie C. Drews, St. Louis, Missouri

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Herpes Zoster Ophthalmicus: Report of Cases and a Review of the Literature. Dr. Ambrose Earl Edgerton, San Francisco, California

---

The Ocular Pathology of Methyl Alcohol Poisoning. Dr. Walter H. Fink, Minneapolis, Minnesota

---

Transscleral Lacrimal Canaliculus Transplants. Dr. Glen Gregory Gibson, Philadelphia, Pennsylvania

---

An Experimental Study of Iridodyalysis. Dr. George L. Kilgore, San Diego, California

---

The Use of Conjunctival Flaps in Cataract Extraction. Dr. Walter Moehle, Brooklyn, New York

---

Objective Strabismometry in Young Children. Dr. Maynard C. Wheeler, New York, New York

OBSERVATIONS ON THE LOCAL USE OF SULFONAMIDES AND TYROTHRICIN IN THE UPPER RESPIRATORY TRACT

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THE PARENTERAL use of sulfonamides in infections of the upper respiratory tract has not been completely satisfactory for several reasons. The occasional serious toxic effect of the drugs has warranted hesitation in prescribing them in adequate amounts in cases where the infection did not appear to be of a severe nature. The infecting organisms have not in some cases been susceptible to the drug, and the ineffectiveness of the sulfonamides in the case of pus filled cavities has limited their value in the treatment of suppurative sinusitis.

The favorable experience with the local application of these drugs in wounds revived the hope of success in their use in infections of the upper respiratory tract. The application of certain of the drugs in powder form in open wounds produced a high local concentration in the adjacent tissue while the blood concentration remained negligible. At the same time the powder exhibited satisfactory bacteriostatic qualities. The only undesirable feature was a fairly marked degree of local irritation.

Conditions in the upper respiratory tract differ in several essentials from those in wounds. The tract is lined by mucosa which for the greater part has ciliated epithelium and is covered by a blanket of mucus which tends to remove any locally applied substance and prevent absorption. The mucosa of the nose and paranasal sinuses has an extensive surface most of which cannot be reached by local medication unless special procedures are used for its introduction into the sinuses. For a satisfactory local therapeutic agent a drug must go into solution in water in sufficient strength for convenient administration, have a bacteriostatic or bactericidal effect upon organisms within the mucosa as well as in the secretions, have no toxic effect upon ciliated epithelium and produce no irritation and/or swelling which might affect the ostiums in such a way as to interfere with the normal removal of secretions.

Sulfathiazole appeared to meet enough of these requirements to justify a trial. During 1940 and 1941 much clinical investigation was carried out on the use of this drug locally in sinus infections and later with its sodium salt.

At the University of Chicago clinics a series of clinical trials was made in cases of acute and chronic suppurative sinusitis. Sulfathiazole was used in the form of a powder insufflated into antrums and frontal sinuses after irrigation, and later it was used in the form of suspensions of finely ground powder in water up to 30 per cent, which was introduced after irrigation and removal of the pus. In some instances a small amount of hydrogen peroxide was also instilled. Improvement seemed to occur in many instances, but the final conclusions were that the results in general were no better than when irrigation only had been used. In many cases

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the recovery of crusts of sulfathiazole weeks after its instillation into an antrum, along with some mucopurulent material, appeared to indicate an undesirable reaction, either irritative or foreign body in type.

Sodium sulfathiazole was also tried in 5 per cent solution both as a nasal spray frequently applied and by introduction into sinuses by irrigation and displacement. While no marked reactions of an unfavorable character were experienced, the solution was subjectively irritating, and treatment was followed by no noticeable improvement or shortening of the course of the infections.

Experience with sulfathiazole, when applied as a dusting powder in sinus cavities after radical operation, has been favorable. It appears to inhibit postoperative infection and may thereby prevent complications.

The powder proved to be most effective when the diseased mucosa had been removed and adequate drainage established, but it does not provide a substitute for either of these two essentials of radical sinus surgery.

TYROTHRICIN

The substance known as tyrothricin was isolated by Dubos[1] in 1939 from cultures of an unidentified soil bacillus and was found to be highly bactericidal for gram-positive organisms.

Later Hotchkiss and Dubos[5] obtained the two crystalline compounds, gramicidin and tyrocidine, from this crude substance. All three substances were found to have bactericidal properties for gram-positive organisms, in vitro and in vivo. Gramicidin is the most effective of the three, but its preparation presents such difficulties that clinical experimen-

TABLE I

Types of Infection	*Number of Cases*	*Average No. of Treatments*	*Method of Application*	*Average Length of Observation*
Acute upper respiratory infection	6	7	Atomizer or nasal instillation (Rose position)	6 to 10 days
Acute suppurative maxillary sinusitis	15	8.6	Instillation through cannula after irrigation with Ringer's	19 days
Acute frontal sinusitis	1	4	"	8 days
Acute suppurative sphenoiditis	1	6	"	14 days
Chronic suppurative maxillary sinusitis	18	16	"	3 months
Chronic frontal sinusitis	4	9	"	4 months
Chronic suppurative sphenoiditis	1	10	"	6 weeks
Chronic ethmoiditis	8	24	Displacement	4 months
Chronic tonsillitis (Hemolytic strep cultures)	8	10	Liberal and repeated applications	1 month
Pathogenic organisms Chronic nasopharyngitis in Nasopharynx	51	15	Postnasal tampon, direct application and displacement by Proetz method	4½ months

tation and treatment of infections has been mainly limited to the use of tyrothricin which has almost the same action although less effective.

Tyrothricin has a hemolytic effect on red blood cells[3,4,6] and is therefore unsuitable for use where it might come in contact with the blood stream. It is not soluble in water and has been proven safe for injection in relatively large amounts[4,7] into the serous cavities of animals and to be effective against certain infections in such cavities. It was used without toxic effects in the cases of human beings affected by surface ulcers, skin infections, postoperative empyema, bladder infection, sinusitis and nasopharyngeal infections.[7] It therefore offered possibilities in the treatment of infections of the paranasal sinuses and the nasopharynx.

CLINICAL INVESTIGATION

A series of investigations was begun in October 1941 embracing the use of tyrothricin in four types of infection, namely, acute infection of the upper respiratory tract, acute and chronic suppurative sinusitis and carriers of pathogenic bacteria in the nasopharynx, pharynx and tonsils. (See Table I.)

Tyrothricin was first prepared for use by dissolving it in alcohol and then diluting the mixture with water to the desired strength. A fine suspension was thereby obtained and the drug was used in strengths of 1:5000 and 1:1000. Propylene glycol was later substituted as a solvent. One gram of tyrothricin was dissolved in 25 cc. of propylene glycol and then diluted with water to obtain solutions of 1:1000, then 1:500 and later 1:100. The highest degree of solubility in propylene glycol was 10 per cent. Precipitation did not occur. Bactericidal properties in vitro were not impaired two weeks after the solution was made up.

The pH of the 1:500 solution was 9.1. This concentration was tolerated well, but the 1:100 solution, which was used later, caused severe irritation in frontal and ethmoid sinuses.

Weaker solutions were used in the first cases but as toxic and irritative effects were found to be absent, more concentrated solutions were used.

Cultures were made from the secretion in all cases before and at frequent intervals during treatment. Red and white blood cells, hemoglobin and urine were checked at intervals. A toxic effect was not noted in any case.

ACUTE UPPER RESPIRATORY INFECTIONS

In this group of six cases treatment was begun on the first to the third day after onset of illness and was carried out daily. Shrinkage of the nasal mucosa with a ¼ per cent solution of neosynephrin was followed by instillation of a solution of tyrothricin with the patient in the Rose position. Solutions of 1:1000 or 1:500 were used. The average period of treatment was seven days.

The course of the infection did not appear to be shortened or alleviated in any case, and the cultures showed no diminution in the growth of organisms as a result of the treatment. (See Table II.)

TABLE II

ACUTE UPPER RESPIRATORY INFECTION CULTURES FROM MIDDLE MEATUS

Organism	*No. of Cases*
Non-hemolytic Streptococcus	5
Streptococcus Viridans	2
Hemolytic Strep.	1
Hemolytic Staph.	3
Staph. Aureus	4
Staph. Albus	2
Pneumococcus	6

Total No. of Cases Treated, 6.

ACUTE SUPPURATIVE MAXILLARY SINUSITIS

This group was comprised of cases of acute empyema of the maxillary sinus which developed after an infection of the upper respiratory tract.

In all cases irrigation with Ringer's solution of the sinus through the ostium was followed by instillation of a solution of tyrothricin through the cannula. Further details of treatment are given in Table III, and the types of organisms cultured are shown in Table IV. While cases in which the condition was mild were excluded from the series, it is obvious that many of those included might have cleared without any irrigation, but they were treated for purposes of investigation. The presence of acute pain was considered to be the criterion for the onset of suppuration. The duration of the infection was definitely not shortened by the use of tyrothricin, nor could it be said that it produced alleviation of symptoms.

ACUTE SUPPURATIVE FRONTAL AND SPHENOID SINUSITIS

In Tables V and VI are outlined the treatment and findings on culture in one case of acute frontal and one of acute sphenoidal suppuration.

TABLE III
ACUTE SUPPURATIVE MAXILLARY SINUSITIS

TREATMENTS				*Concentration of Tyrothricin*			*Average Duration of Empyema*	
No. of Cases	No. of Sinuses	Frequency	Average No. per sinus	1:5000	1:1000	1:500	Before therapy	During therapy
15	23	Every 1-7 days	8.6	2 cases	12 cases	1 case	2 days	19 days

TABLE IV
ORGANISMS CULTURED FROM ACUTE SUPPURATIVE MAXILLARY SINUSITIS

Organisms	*No. of Sinuses Infected*
Strep. Hemo.	4
Non-Hemo. Strep.	10
Strep. Viridans	16
Staph. Aureus Hemo.	12
Staph. Aureus	13
Staph. Albus	14
N. Catarrhalis	22
Pneumococcus	17
B. Influenzae	2
B. Coli	1
Mucococcus capsulitis	1

Total: Species of organisms——11; No. of cases treated, 15; No. of sinuses treated, 23; No. of cultures taken, 121.
Note. — All cases had mixed infection at some stage. Cultures varied from day to day similar to those shown in Figure VI.

Irrigation of the sinus was not begun in either case until the acute reaction of the nasal mucosa had subsided. The sphenoid ostium was cannulated easily in each instance, while the frontal sinus was entered into with slight difficulty. In the latter case there was some aggravation of symptoms after treatments. Neither the course of the disease nor the cultures demonstrated any favorable effect from tyrothricin.

CHRONIC SUPPURATIVE SINUSITIS

The investigations in chronic suppurative sinusitis included cases of disease of the maxillary, frontal and ethmoidal sinuses. (See Table I.) The course of treatment for the cases of maxillary sinusitis is shown in Table VII, and the cultures made from the secretions are shown in Table VIII. The cases of frontal sinusitis were also treated by irrigation and instillation per cannula, and the cultures were

TABLE V

TYROTHRICIN THERAPY IN ACUTE SPHENOID AND FRONTAL SINUSITIS

	Sphenoid	*Frontal*
No. of Cases	1	1
No. of Treatments	6	4
Method of Treatment	Irrigation through ostium. Instillation of Tyrothricin through cannula.	Same
Concentration Tyrothricin	1:5000-1:1000	1:1000
No. of Cultures	6	4
Clinical Result	Tyrothricin ineffective as adjunct to shrinkage and irrigation.	Local reaction and aggravation of symptoms.
Effect on Cultures	None demonstrated.	None demonstrated.

TABLE VI

BACTERIOLOGY IN ACUTE SPHENOID AND FRONTAL SINUSITIS
GROWTH OF ORGANISMS IN EACH OF THE CULTURES

Organisms	*Cultures from Sphenoid*						*Cultures from Frontal*			
	1st	2nd	3rd	4th	5th	6th	1st	2nd	3rd	4th
Hemolytic Strep			+	+	++	+	++	+		++
Non-Hemolytic Strep	++	+		++		++	+	+++	+	
Strep Viridans	+	++	++		++			++	++	
Pneumococcus	+++	+++	++	+++	+++	++				+
Staph. Aureus			+	++	++	+++	+++	+	+++	+++
B. Influenzae					+		++			

Note. — Some of the cultures were placed in Rosenow's brain broth for 18 to 20 hours and then plated on blood agar. Some were plated directly on blood agar. Anaerobic cultures were made on a small series which is not included in the tables. The variation from time to time was typical of all cases of acute suppurative sinusitis. Contamination from the nose was unavoidable in most cases.

TABLE VII

CHRONIC SUPPURATIVE MAXILLARY SINUSITIS

Number of cases	*Number of sinuses*	TREATMENTS			*Concentration of Tyrothricin*		
		Frequency	*Average number per case*	*Method*	1:10000	1:500	1:1000
18	27	Every 2 to 7 days	12	Irrigation with Ringer's followed by Tyrothricin per cannula	2 cases	5 cases	11 cases

comparable. The cases of ethmoidal sinusitis were treated by shrinkage and displacement by the Proetz method.

While gram-negative organisms were more common in the group comprising the cases of chronic sinusitis, the gram-positive organisms, which are susceptible to tyrothricin, predominated here as in the acute infections.

The instillation of tyrothricin in these cases of chronic suppuration and empyema produced no improvement over the customary conservative treatment by irrigation.

I) was comprised chiefly of patients with chronic arthritis in whom a focus of infection was suspected. Chronic inflammatory changes were evident in some, while in many, local symptoms and signs of inflammation were slight or absent.

These patients were treated by three methods. Some were treated by means of postnasal tampons, some by instillation of the drug through the nose while in the Rose position and some by the latter method plus displacement. Treat-

TABLE VIII

CULTURES IN CHRONIC SUPPURATIVE MAXILLARY SINUSITIS

Organisms	*No. Cases Infected*	*No. Sinuses Infected*	*No. Positive Cultures*
Hemolytic Streptococcus	3	4	16
Non-Hemolytic Strep.	7	11	28
Streptococcus Viridans	12	18	39
Hemolytic Staph. Aureus	9	16	27
Staph. Aureus	13	17	52
Staph. Albus	11	13	23
N. Catarrhalis	17	25	46
Pneumococcus	16	21	51
B. Influenzae	2	2	5
Friedlanders	5	5	16
B. Coli	1	1	2

Total No. case treatments..... 230
Total No. sinus treatments..... 339
Total cultures from all sinuses..... 181

CHRONIC INFECTION IN THE TONSILS, PHARYNX AND NASOPHARYNX

The eight patients with chronic tonsillitis (see Table I) were carriers of hemolytic streptococci. Daily treatments by liberal and repeated application of a solution of tyrothricin for an average of six days failed to produce negative cultures.

The large group with pathogenic organisms in the nasopharynx (see Table ments were given every two to seven days. The bacteriology is summarized in Table IX. The cultures showed considerable variation even when taken every two or three days, but no significant change was found as a result of the treatment. While the majority of these patients claimed improvement, it proved to be temporary and was not borne out by any change in local appearance or on culture.

TABLE IX
CHRONIC NASOPHARYNGITIS

Organisms	Number of Cases	Total number of positive cultures	Chart of Successive Cultures in One Typical Case										
	Total number of cases treated: 51	Total number of cultures made: 561	1	2	3	4	5	6	7	8	9	10	11
Hemolytic Strep.	12	37					+	+			+		
Non-Hemolytic Strep.	37	197				+ +			+ +				
Strep. Viridans	43	343	+		+	+ + +	+			+	+ + +	+	+ +
Hemolytic Staph. Aureus	13	48							+	+ +		+ +	
Staph. Aureus	25	109				+		+ +			+		+ + +
Staph. Albus	26	82		+ +		+						+	
Pneumococcus	47	381	+ +		+ + +		+ +		+ + +			+ +	+
Neisseria Catarrhalis	43	412	+ +		+	+ +	+ + +	+	+ + +		+ +	+ + +	+ + +

SULFADIAZINE SPRAY

Following the negative results with the solution of tyrothricin a spray consisting of a 2½ per cent solution of sulfadiazine was tried in two types of cases; the acute infection of the upper respiratory tract and the carriers of pathogenic bacteria in the nasopharynx.

The spray consisted of a solution of 2½ per cent sulfadiazine and 8 per cent triethanolamine in water. The pH could not be brought below 8.2 without causing precipitation.

The patients with acute infection of the upper respiratory tract were treated with the nasal spray from four to eight times daily. No improvement could be determined as a result of the spray either in the duration and severity of the infection or in the growth of bacteria on culture.

The patients with nasopharyngeal infection were treated both by nasal spray and by daily displacement in a few instances to obtain contact for a longer period of time. Considerable discomfort resulted from the latter treatment probably due to the high pH of the solution.

The culture controls showed no definite improvement as a result of this treatment.

DISCUSSION

Investigations with sulfathiazole, sulfadiazine and tyrothricin as local therapeutic agents administered in cases of infection of the upper respiratory tract have proved to be definitely disappointing except when used locally following radical surgery.

Whether the extract of penicillin discovered by Fleming[2] will prove to be effective in the local treatment of the upper respiratory tract remains to be determined. The fact that it may be suitable for parenteral as well as local use seems to promise more success.

The characteristics required of any local therapeutic agent for success include:

1. Sufficient bacteriostatic and bactericidal action in the presence of pus and mucus.
2. Non-irritating and non-toxic to nasal and sinus mucosa.
3. Sufficient absorption into the mucosa to combat infection.
4. No interference with normal defense mechanisms of the mucosa.

In addition to these essentials, there is the problem of getting sufficient contact with the mucosa in acute infections, and of dealing with the permanent mucosal changes in chronic infections which predispose to reinfection. In the latter type of case it is probably unreasonable to expect a local chemotherapeutic agent to act as more than an adjunct to establish procedure for drainage.

While the search for a satisfactory local therapeutic agent has been so far disappointing, the parenteral use of sulfonamides has proven of great value in certain types of infections of the upper respiratory tract. Their value has been demonstrated in severe acute sinus infections and their complications, in carriers of hemolytic streptococci following acute infections and in preventing postoperative infection following sinus surgery. While the serious complications from the use of sulfonamides are frequent enough to make their indiscriminate use definitely unsafe they provide most effective protection in severe infections.

The maintenance of sufficient concentration of the drug in the blood in severe acute sinus infections appears to be the most adequate way of keeping the infection localized. The drug has little effect upon an empyema in a sinus cavity, and may have to be combined with removal of the pus for the best results.

The undesirable effect of trauma to the mucosa and the danger of promoting a spread to deeper tissues and bone have weighed against removal of pus in acute sinus infections in the past. Now, however, the maintenance of an adequate concentration of sulfonamide in the blood prevents a spread of infection within the tissue and eliminates the chief element of danger from removal of the pus in those cases where severity of symptoms and signs indicate the necessity for its removal.

While the use of adequate sulfonamide therapy decreases the need for any nasal interference, it also provides valuable protection in those fulminating cases where drainage of pus is desirable. Trauma to nasal mucosa in the region of ostiums remains an important consideration in the choice of a method of drainage. In the case of the maxillary sinus, puncture may be preferable to trauma in the middle meatus. In the severe or fulminating frontal sinus suppuration, the external drainage through a trephine opening remains the preferable method and with the use of sulfonamides can be done without fear of consequences from trauma to the bone.

SUMMARY

The requirements for a satisfactory local therapeutic agent for use in the upper respiratory tract have not been met either by any of the sulfonamides or by tyrothricin.

The greatest advance in the treatment of severe acute sinus infections has been the parenteral use of sulfonamides in adequate concentration. By this means the spread of infection is limited, and any necessary surgical procedures may be carried out in much greater safety.

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DISCUSSION

THEODORE WALSH, M.D., St. Louis, Missouri: I was very happy to read Doctor Lindsay's paper and to hear him present it, because he has given a thoroughly objective and carefully controlled series of cases in which the effects of the local use of tyrothricin and of the sulfonamides in infections of the upper respiratory tract have been determined.

At Washington University School of Medicine tyrothricin was tried in the prevention and treatment of infections of the upper respiratory tract, and the results obtained were the same as those which Doctors Lindsay and Judd have reported.

Doctor Hartmann of St. Louis attempted to determine whether there was any alteration in the flora of the nasopharynx in children following the use of tyrothricin in 1:500 solution. As a matter of fact, I think that "solution" is the wrong word; the mixture is a finely divided "suspension" of tyrothricin. He found no changes in the flora. It was then thought that perhaps the reason satisfactory results were not obtained was because the drug itself did not come in contact with organisms that were perhaps deep in the tissues so tyrothricin was placed in suspension in propylene glycol. The drug is soluble providing saline is not added.

This suspension was used by us (we called it T.P.G.) in an effort to prevent colds in a group of children at Ridge Farm, a convalescent home for rheumatic children. As controls, saline and vaccine were used. The group of children who were given tyrothricin suspended in propylene glycol had more colds than any of the others, even more than those who were given only normal salines. I believe that the reason for this is easily understood. In chronic infections the organisms are not on the surface; they are down deep in the tissues, and the suspensions that were used did not reach these organisms at all. Certainly, if the drug is absorbed to some degree, it is not present in sufficient quantities to be of any value.

We were disappointed with tyrothricin. I was very much interested to hear Doctor Lindsay's comments, because there have been some enthusiastic reports concerning the local application of all the sulfonamides and of tyrothricin.

In the acute infections T.P.G. was used and the conditions of our patients became definitely worse. The irritation was very uncomfortable, and the acuteness of the infection lasted two or three days longer than usual. In only one case was there any evident value and that was in a woman with acute pharyngitis; she sprayed her throat with T.P.G. three or four times a day and claimed immediate relief. I think that the sore throat was probably psychologic rather than pathologic.

As far as the sulfonamides are concerned, I believe that the introduction of the various sulfonamides into the sinuses is not devoid of danger. If one puts a sulfonamide in powder form into a sinus, it remains there as an inert foreign body. It is interesting to note that ciliary function is not destroyed by the presence of this powder. I tried it in some animals first. I opened one rabbit's antrum and filled it with sulfathiazole, and in another rabbit I used sulfapyridine. About a month later the animals were killed, and their antrums were opened. There was a mass of the drug, just like a stone, lying in the sinus. I removed the mass, and the cilia were beating well under it. This happened in dogs as well as in rabbits. I do not believe that the sulfonamides can be placed in solution in sufficient concentration to affect organisms that are deep in the tissues.

I thank Doctor Lindsay for this excellent paper.

LAWRENCE R. BOIES, M.D., Minneapolis: One seldom hears or reads a medical paper that reports negative results. Too often we are informed of some therapeutic proposition which has not been adequately tested but over which some one has become prematurely enthusiastic. If the treatment relates to such popular maladies as head colds or "sinus trouble" or allergy, it is certain to excite a

general interest, to be featured in the newspapers or called to our attention by our patients who have read about it in the *Readers Digest*.

Doctor Lindsay has made this study in his usual thorough manner. The only criticism that those who disagree with him might make is that the number of cases studied is small.

At the University Hospital at Minnesota, we have used the sulfonamides and tyrothricin locally in a variety of ear, nose and throat conditions. We are convinced that local application of these substances in solution to the mucosa of the nasal space, and to the pharynx, has no special merit in acute or chronic inflammations. We have found that the local use of a sulfonamide powder postoperatively in radical surgery for chronic suppuration seems to lessen secondary infection. In surgery for acute disease, such as mastoiditis, we have discontinued its use in the wound except in the rare instance where there has been early intervention. If one cleans out the mastoid thoroughly in the coalescent type of the disease, healing is rarely a problem.

Doctor Lindsay has indicated that the parenteral use of the sulfonamides makes certain surgical procedures safe in acute suppurative sinusitis. I have not had occasion to test this. It is my belief that no sinus needs surgical drainage in the acute phase of the infection except the frontal sinus and then only when there is evidence of continued tension within the sinus. A simple trephine through the floor of the sinus has in my experience been a safe procedure. However, the patients on whom trephines have been performed in the past three or four years have all had sulfonamide therapy, and this may have been a factor in our good results.

Doctor Lindsay has also indicated that the parenteral use of the sulfonamides has proven to be effective treatment in the cases of carriers of hemolytic streptococci. This has been contrary to our experience at the University Hospital where this has been evaluated by Doctor Wesley Spink.

I wish to thank Doctor Lindsay for his report. When I read his paper I was reminded of a statement which Sir William Osler is said to have made: "In treatment, the placid faith of the believer, not the fighting faith of the aggressive doubter, is our besetting sin."

Doctor Lindsay lacks that placid faith.

HENRY L. WILLIAMS, M.D., Rochester, Minnesota: Doctor Lindsay's criticism of the present tendency to report semi-magical results from use of some new or obscure drug which seem based on wishful thinking rather than clinical observation and careful bacteriologic and pathologic checking is valuable. As the previous discusser has stated, there is great need of such papers.

At the Mayo Clinic we have been trying to assess the value of gramicidin for the past three years, but I do not feel that we have reached a state of knowledge at which a report can be given.

The work of Herrell and Heilman indicates that about four hours are required for a complete bactericidal action of either gramicidin or penicillin to occur. I doubt very much, therefore, that spraying the membranes with a 1:1000 suspension of crude gramicidin (tyrothricin) could be expected to be of much value. Certainly this material will not cling to the membranes for a period of four hours. Another point which should be made in a discussion of this type is that gramicidin is a bactericidal substance only and cannot be expected to rejuvenate damaged tissues or abolish those physical malformations which are so often present in cases of chronic sinusitis.

It should be clear from the start that gramicidin could be effective in only a carefully selected group of cases in which the bacteriologic, pathologic and anatomic conditions were such that the preparation could be expected to work. Because of these facts and because criteria for selection of patients for treatment had not been established little of the work presented in this paper can be supposed to furnish a test of the bactericidal effect of gramicidin.

Of considerable importance are the bacteriologic studies carried out in these cases, because gramicidin is not as effective against staphylococci as are penicillin or such newer preparations as the quaternary ammonium compound, phemerol.

In my own group of cases I have tried to select cases of chronic sinusitis in which chronic changes were not so far advanced as to appear to be irreversible, in which anatomic anomalies which would tend to rapid re-infection were not present and in which bacteriologic studies seemed to warrant a trial of the agent.

I have treated 16 patients who seemed to be suitable as judged by the criteria which I have given. It is my impression when looking over my notes that the response of about half of them was good to treatment with gramicidin and that of the other half could not be

considered any better than fair or poor. I am still trying to establish a basis for clinical selection which I do not believe can be done in a hurried and superficial manner.

Doctor Lindsay's remarks in regard to the use of sulfonamide preparations are obviously correct, as the conditions under which their use often has been advised are an impossible test of their therapeutic value. I think the essayist is to be congratulated on this attempt to discourage enthusiastic reports on methods of treatment which a little reflection indicates to be worthless.

J. R. LINDSAY, M.D., Chicago, Illinois: I want to thank the discussers for their remarks. I did not know whether their experiences would coincide with mine or not.

There is no question about the bactericidal action of gramicidin on gram-positive organisms. Under the peculiar conditions with which one has to deal in the upper respiratory tract and paranasal sinuses, however, the substance has not, in our experience, proven of therapeutic value.

THE EFFECT OF EXPLOSIONS ON THE ACOUSTIC APPARATUS

H. B. Perlman, M.D.
CHICAGO

War has again aroused interest in the subject of acoustic trauma because more and more people are exposed to the great noises associated with modern warfare. The common, but poorly understood, source of noise in war is the explosion. The term "explosion" is used to describe the phenomena that occur when a solid or liquid is suddenly changed into a gas. Usually this change takes place within 1/100,000 of a second or less. The molecules of the air are compressed by this abrupt change in volume of the explosive, and this molecular compression is followed at once by a much slower phase (lasting 1/1000 second) in which normal molecular pressures are regained

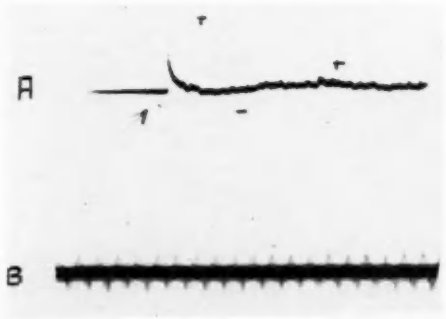

Fig. 1 — A. Record of the pressure to time relations of a shock pulse from a powder blast.
B. 1000 cycle timing wave.

and by a still slower and much less intense phase of negative pressure lasting 1/100 second (fig. 1). Near its source, the molecules are forced outward for a considerable distance before oscillating. Beyond this the wave is propagated like a sound wave from molecule to molecule, each molecule oscillating about its resting position. Ordinary sound waves are characterized by an equal average of molecular compressions and rarefactions. To be heard, these periods must last longer than 1/20,000 second but less than 1/20 second. The peculiar pressure wave initiated by an explosion is called a shock pulse. A shock pulse may be defined as a sound wave of great condensation and great initial velocity. The war is literally fought with these shock pulses. Their intensity is inconceivably greater than the intensity of ordinary sound waves. This enormous force of the shock wave is used in various ways. Generated by the explosive in a bomb it can itself destroy the machines of war, even under water. The shock pulse generated by a 4000 pound bomb can blow down a square block of buildings and shatter glass windows for several blocks beyond that. The shock pulse generated by gunpowder is used to propel a bullet anywhere from 1000 feet to 25 miles. Used in the bullet itself, it is made to shatter the shell casing into fragments after it arrives at its target. The shock pulse generated by exploding gasoline or oil is used to drive the airplane, tank and marine engine. The shock pulses generated in the cylinder head of an engine find their way out through the exhaust pipe. The great destructive power of the shock pulse is in part due to the sudden enormous rise in temperature that results in the development of great pressure. Near its source the velocity of the pulse due to its high pressure is greater than that of sound — that is, greater than 1100 feet per second. As the shock pulse or

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From the Division of Otolaryngology of the University of Chicago.

wave is propagated in air it undergoes a change in shape as well as a reduction in intensity because of the resistance of the air molecules to such profound compression. There are great temperature fluctuations in each cycle, and the intensity of the pulse falls off more rapidly than the inverse square law for ordinary sound. In water the shock pulse is propagated with less loss of energy than in air. Nine ounces of guncotton exploded under water can be definitely recorded under the sea at 40 miles.[1] If the ear is submersed it might be exposed to trauma from this source. Variations in the shape of the shock pulse may also occur at its source by altering the character of the explosive. Gun powder used to propel the bullet burns more slowly (a few thousandths of a second) than T.N.T. used to explode the bullet (10 millionths of a second). Their pressure waves are distinguished by differences in abruptness and duration. One of the effects of this alteration in the shape of the shock pulse is a change in its sound. But according to Steudel, the shape of the pulse wave up to .3 milliseconds has little to do with the sensation of loudness. The only reason why the ear responds to the shock pulse at all and produces an auditory sensation is because it occurs within a period of time to which the auditory mechanism is normally made to respond. The ear can be considered as a mechanism made to respond to changes in pressure occurring within certain time intervals. If this time interval is slower than 1/20 of a second or faster than 1/20,000 of a second, no localized disturbance of the basilar membrane is produced. Hence there is no irritation of the end organ and no auditory sensation. The conducting mechanism and the cochlea will respond to a single pressure disturbance as well as to a succession of such disturbances. When the auditory mechanism is exposed to regularly recurring symmetrical pressure pulses 1000 times a second, it is sensed as a 1000 cycle pure tone. If the ear is exposed to but a single asymmetrical pressure wave of a shock pulse lasting 1/1000 second a localized irritation of the end organ is also produced but the sensation is described as a "bang" or "crack." When the rise in pressure is very abrupt and total duration short, as when near a source of explosion, the auditory sensation may be described as a sharp crack. As the shock pulse is propagated and loses its intensity and abruptness and is reflected from many objects along its path, the auditory sensation changes to a dull boom or rumble. All of us have experienced these differences in auditory sensations during a thunderstorm. Lightning or an electric spark is a common source of the shock pulse. When one is close to it, the auditory sensation resulting from the shock pulse called thunder is described as a sharp crack. When one is further away from the source of lightning, reduction in size of the pulse, reflected pulses and various times of arrival of the pulses at the ear cause the low rumbling sound of thunder. Similarly gun fire may be heard as a sharp crack when nearby and as a dull boom when far away. Secondary acoustic oscillations set up in different sized gun barrels are also important in the sound of a gun. The pressures in any of the shock pulses regardless of the sound they make may nevertheless be great enough to produce gross ear injuries such as rupture of the ear drum and damage to the conducting mechanism, just as the relatively slow pressure change of a slap on the ear may do the same. Static pressure of 200-400 mm. Hg. will also rupture the drum.

Knowledge of the physical properties of the ear is important in understanding its response to a shock pulse. The conducting mechanism of course cannot follow a pressure rise of 1/100,000 second but attempts to. The initial displacement of the mechanism therefore is very

rapid, corresponding to a sound wave of high audible frequency. This probably produces a localized stimulus near the basal end of the cochlear duct and is heard as a high pitched crack sound. The major disturbance set up in the conducting mechanism by the shock pulse consists usually of a single asymmetrical but fast positive and negative wave. This oscillation is many times larger than any of the associated secondary waves and is the important traumatizing factor for the ear. The entire vibration may last about 1/1000 of a second (fig. 2). The greater negative wave of the induced oscillation is in part due to the longer exposure to this part of the shock pulse and in part to the fact that for a given amount of pressure, movement outward of the malleus is greater than movement inward. The lower natural frequency of the conducting mechanism probably works to reduce progressively the speed of the oscillation of the ossicular chain. Also the large natural damping qualities of the conducting mechanism prevent a prolonged response to a single shock pulse. The natural frequency and damping of the middle ear mechanism is found by displacing the mechanism within its elastic limits and by allowing it to fall back of its own accord to a resting state.

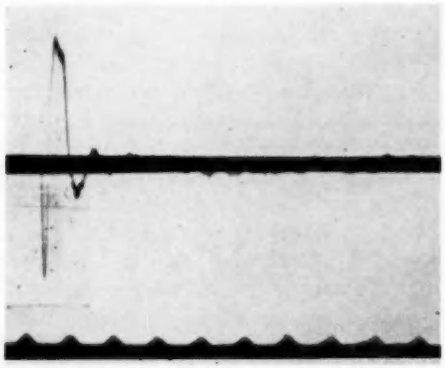

FIG. 2 — A. Record of response of the human conducting apparatus to a shock pulse, light point reflected from a mirror on head of the malleus.
B. 1000 cycle timing wave.

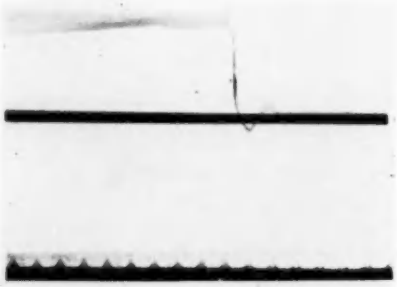

FIG. 3 — Record of the resonance frequency and damping characteristic of the human ossicular chain in a fresh cadaver ear. The chain was displaced by a fine hook (within its elastic limits) and allowed to fall back to rest in its own manner. The resonance frequency is about 700 cycles per second. The high degree of damping prevents a prolonged reaction of the ossicular chain when stimulated by a shock pulse. Timing wave — 1000 cycles.

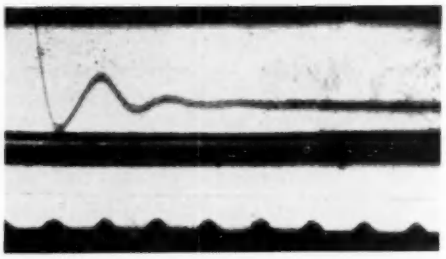

FIG. 4 — Same as figure 3 but mechanism displaced beyond its elastic limits shows hysteresis effect. Timing wave — 1000 cycles.

I hope that the above brief description has indicated to you how the shock pulse acts upon the ear to produce an auditory sensation. Now a word about the amount of pressure developed by the pulse. To gain proper perspective we may review the sensitivity characteristics of the ear to ordinary sustained pressure waves called sound. At 1000 cycles per second the ear can detect a sound wave whose energy flow is 10^{-16} watts per sq. cm. or whose pressure is about one five billionths of an atmosphere. When sustained sound reaches a pressure level of 10^{-4} watts or 120 decibels above threshold it becomes unbearably loud. The intensity of a shock

pulse is usually much greater than that found in the loudest sustained sound. The pressures developed may be many atmospheres. Thompson,[10] a ballistic expert at the naval proving ground, measured the intensity of a shock pulse 26 feet from the explosion of 10 pounds of T.N.T. and found it to be 10^4 watts per sq. cm. That is 80 decibels above the threshold of feeling or 200 decibels above the threshold for hearing of sustained periodic sound. Naturally the closer to the source, the greater the energy, especially with a shock pulse whose intensity falls off more rapidly than the inverse square law. Besides the shock pulse from the powder charge, two additional shock pulses are produced in gun fire (fig. 5). These two pulses originate from the nose and base of the bullet because it plows through the air faster than the velocity of sound. Miller[6] made records of these pulses during the last war, and these records serve to illustrate what happens when any gun is fired. A representative observation taken from a station located 600 feet in front and 300 feet to the side of a 12 inch gun can be described. Sound effects called the "crack" and "boom" of the gun are recorded (fig. 6). The "crack" comes from the projectile and is due to two sharp compression pulses about 2/100 second apart with a negative pulse between them. These pulses are due to shock pulses developed at the point and base of the projectile. The "boom" sound is due to the shock pulse which originates from the exploding gas suddenly "uncorked" at the muzzle of the gun by the bullet and at this distance occurs 1/10 second after the "crack" and lasts 15/1000 second. At this distance from the gun the pressures of these pulses are all about equal or about 1.5 gm. per sq. cm., much greater than ordinary sound pressures. At this distance from the gun the crack and boom occur in too rapid succession to be differentiated by the ear. The differences in their shape explain the differences in the auditory sensation they produce when they are heard separately as when an observer is one-half mile in front of the gun. The crack is heard first because it travels towards the observer

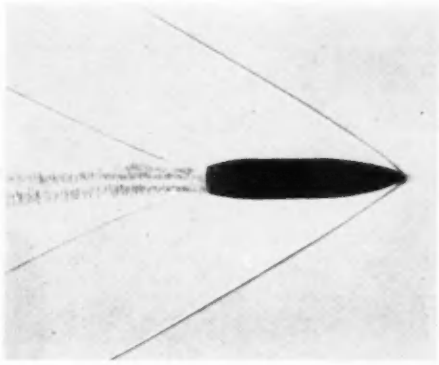

FIG. 5 — Sound waves produced by a bullet moving with a velocity of 2,700 feet (823 meters) per second. These waves from the nose and base of the bullet cause the "crack" heard by an observer stationed at some distance from the gun and are recorded in the first part of the tracing in figure 6. From Miller6 by permission of The Macmillan Company, publishers.

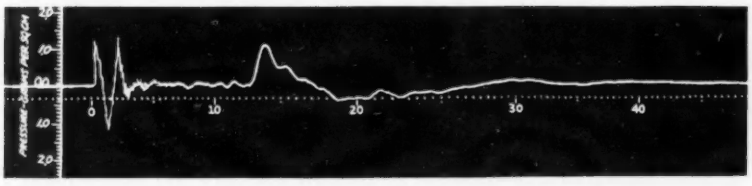

FIG. 6 — Phonodeik record of the shell and gun waves from a 12 inch (30.5 cm.) rifle. Two positive pulses immediately after zero time, with a negative pulse between them, come from the nose and base of the shell and are heard as a "crack." The slower pulse after 1/10 second comes from the exploding powder in the gun barrel and is heard as a "boom." This record was taken from a position 600 feet (183 meters) in front of and about 300 feet (91.5 meters) to the side of the gun. From Miller6 by permission of The Macmillan Company, publishers.

with an initial velocity equal to that of the moving projectile (2600 feet per second), more than twice the velocity of sound. The boom traveling with the mean velocity of sound (1100 feet per second) arrives at the ear later — when 3000 feet in front of the gun — about one second after the crack and is heard separately.

Miller also determined the pressures prevailing at various points near a discharging gun. In general, any position in front of the muzzle of the gun is dangerous because the shock pulse comes from there. The maximum pressures are recorded in front of the muzzle varying with the elevation and its size; i.e., 542 pounds (45 atmospheres) or 38,200 gm. per square inch on the ground 12 feet in front of a 12 inch gun. While the pressures about the breach of the same gun, where the men are stationed, are much smaller, for example, 100 gm. per sq. cm. and less, they are enormous when translated into ordinary acoustic pressure equivalents. When one is close to a gun he cannot hear separately the three shock pulses but the ear nevertheless is exposed to their trauma. When small firearms are used for target practice in a bare walled room, these shock pulses are reflected from the walls and thereby further expose the ear to trauma (fig. 7).

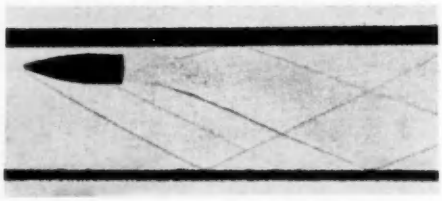

FIG. 7 — Reflection of shock pulses from a bullet by bare walls (after Cranz).

The shock pulse of a .32 calibre blank cartridge a foot from the external canal produces a violent displacement of the ossicular chain experimentally on a fresh temporal bone (fig. 8). A comparable displacement can be obtained by 2 cm. of water static pressure in the external canal. Translating this into acoustic pressure it is very great. If one tries to drive the same mechanism with a 1000 cycle tone of 100 decibel intensity through tubing sealed in the external canal he cannot get a visible disturbance with the same optical magnification.

What means does one have of protecting himself from these shock pulses? The ear is partially protected from these shock pulses by its physical inability to follow faithfully a pressure change occurring within 1/100,000 second. Another effective natural protection of the ear to great forces of positive pressure is found in the incudo malleolar joint.[9] For sustained sound of moderate intensity, the malleus and incus rock as a unit around the axis extending from the fan shaped ligament of the incus to the anterior ligament of the malleus. There is no differential motion in the joint. But this joint is not fixed when the inward motion of the malleus is large. When there is great inward motion of

FIG. 8 — Response of the human ossicular chain (malleus) to the shock pulse from a blank cartridge. 1000 cycle timing wave. Positive pressure phase of oscillation is toward the timing wave.

the malleus, dislocation occurs at this joint that prevents transmission of the motion of the malleus to the incus. This does not hold true for forces of negative pressure moving the malleus outwards. Here the incus and malleus motion is essentially as if the incudo-malleolar joint were fixed. But extensive outward displacement of the malleus may be checked by the tonus of the tensor tympani muscle. Another factor which prevents excessive displacement of the cochlear fluid to large acoustic stimuli is the changing axis of motion in the stapes footplate. Moderate motion of the incus forces the stapes to pivot about its posterior pole. The annular ligament is wider at the anterior pole. This is where the greatest movement takes place. Greater motion of the incus causes the stapes to rock about the long axis of the footplate, and this reduces the resulting movement of fluid because of the limitations of the annular ligament along the upper and lower margins of the stapes.

The ear has another normal protective device in the acoustic contractions of the middle ear muscles. But a certain time must elapse between the moment the sound wave strikes the ear and initiates a disturbance in the cochlear nerve, and the moment this disturbance reaches the motor nerves that innervate these muscles. In man, the latent period for the acoustic contraction of the stapedius muscle is 10.5 milliseconds,[7] and it is almost certain that the latent period of contraction of the tensor tympani is about the same (fig. 9). Unfortunately, the shock pulse is a much shorter phenomenon. Many traumatizing noises are exceedingly short, i.e., those produced by riveting, punch presses, drop forges, chipping steel, telephone receiver bangs, etc. The major disturbance of the conducting mechanism and the end organ produced by a shock pulse may last about 1/1000 second. Hence the middle ear muscles can play no part as a protection against the shock pulse. The auditory mechanism may be exposed to about 10 pulses of this kind before the muscles begin to act. Rapid firearms are particularly bad in this respect. On the other hand the location of the ear with respect to the source of the shock pulse is important because the head is a very effective shield for these pulses. The ear facing the shock pulse may be the only one damaged or at least is more exposed to injury than the ear on the opposite side of the head. Men who fire a gun from the right shoulder place their head in such a way that their left ear faces the muzzle where the shock pulses originate. Hence their left ear suffers more damage than the ear on

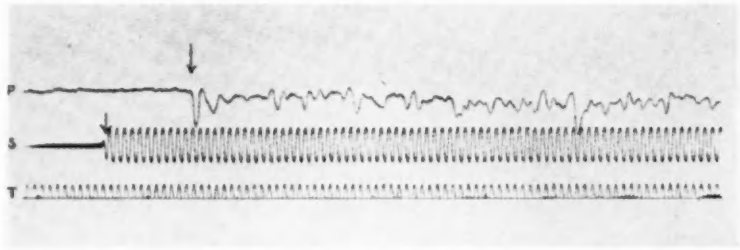

FIG. 9 — Record showing latency of the acoustic stapedius reflex contraction in the right ear of Mr. C. At T a 1000 cycle timing wave. At S the sound stimulus coming from an open tube 2 cm. from the left external auditory meatus, and beginning at the point indicated by arrow. At P the physiological track showing the abrupt onset and sustained character of the action potentials. An arrow indicates the onset of these potentials. The latency period is the time interval between the sound onset (see arrow) and the onset of the action potentials (see arrow).

the opposite side, shielded by the head. Distance from the source and the exact position of the exposed ear with respect to the source are both important factors influencing the intensity of response in the ear (figs. 10, 11, 12, 13). If the external canal directly faces the oncoming wave or is at an angle to it, alterations in the response occur (figs. 14, 15, 16, 17). There are many ways of shielding the head as well as the body from the shock pulse. Any wall or partition will act as a shield. A mobile steel plate braced against the ground can shield a man from enormous explosions. Lying in a depression in the ground or even

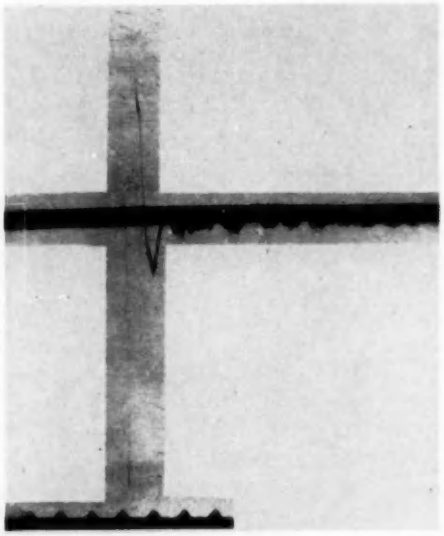

FIG. 10 — Large oscillation of the malleus to a shock pulse at the ear. Compare with figure 11. Timing wave — 1000 cycles. Positive pressure towards timing wave. NOTE: Top of negative wave not visualized (beyond film margin).

FIG. 12 — Reduced oscillation and change in wave form. Shock pulse 20″ away from external auditory meatus. Compare with figures 10 and 11. 1000 cycle timing wave.

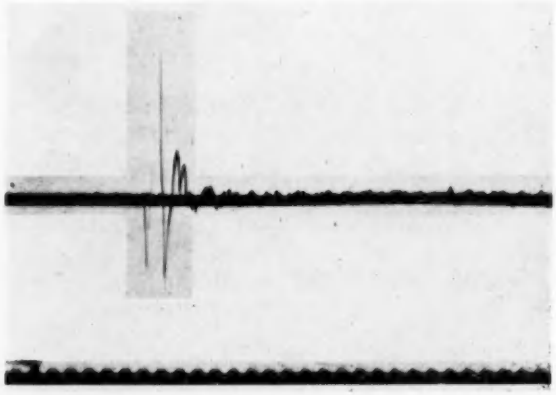

FIG. 11 — Reduced oscillation of malleus and change in wave form. Shock pulse 15″ from external auditory meatus. Compare with figure 10. 1000 cycle timing wave.

lying flat on the ground is effective because when a shock pulse originates from a bomb or shell striking or entering the ground the greatest energy of the pulse travels upwards and not along the surface of the earth. Putting a shield on a field piece or a turret around a naval gun protects the men from the shock pulses originating at the muzzle. Protection to the ear itself may be obtained by closing the ear with the finger, wearing a helmet covering the ear or using some type of ear plug (figs. 18, 19, 20, 21). Many effective obturators have been developed and tried but all have their shortcomings. A deep helmet covering the ear may be an adequate shield for the shock pulse without interfering with the sustained speech sounds. Precautions should be taken especially in training but they are sometimes difficult to carry out in the heat of battle. Covering or plugging the ear deafens it for ordinary sound and one's life may depend on his ability to hear commands and sense danger. But conversely deafening the ear by the noise of the shock pulses may interfere with the ear's ability to hear signals and commands. A deep helmet shielding the external auditory meatus may be the best answer for the infantry.

A single exposure to a shock pulse may permanently deafen the ear. The

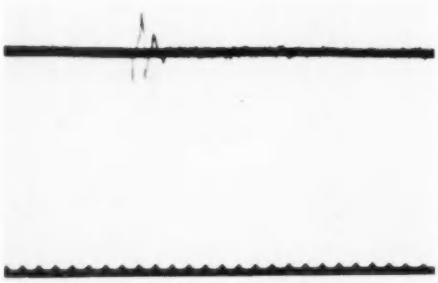

FIG. 13 — Marked reduction in oscillation of malleus. Shock pulse 36" away from the external auditory meatus. Compare with figures 10, 11, 12. 1000 cycle timing wave.

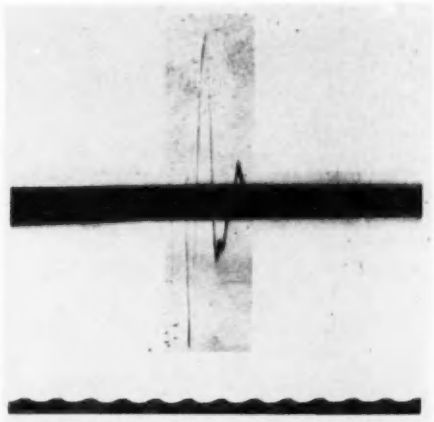

FIG. 14 — Oscillation of malleus to shock pulse, external auditory meatus facing the wave front (gun fired at the ear). Compare with figure 15. 1000 cycle timing wave.

FIG. 15 — Oscillation of malleus reduced — external auditory meatus at an angle to wave front of shock pulse (gun fired from behind the ear). Compare with figure 14. 1000 cycle timing wave.

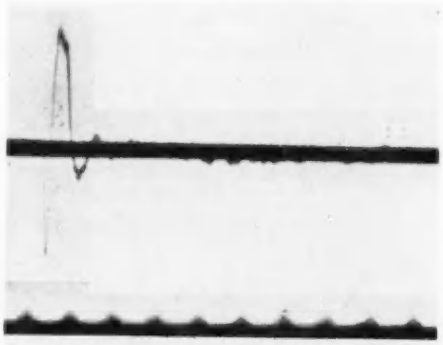

FIG. 16 — Oscillation of malleus when external canal faces shock pulse. 1000 cycle timing wave.

audiogram is rather typical — good hearing for low tones and an abrupt high tone loss for both air and bone conducted sound. In the early stages only an island of deafness is found in the audiogram between 4000 and 6000 cycles (fig. 14). This can be produced though not regularly by the shock pulse from a .32 blank cartridge. Discharged three feet from the ear, recovery occurs in 30 minutes* (figs. 22, 23). The upper tone limits as tested with a monochord may be normal. This type of

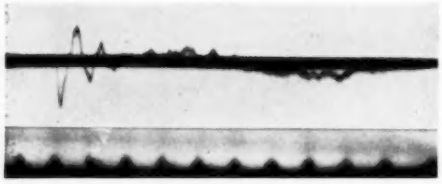

FIG. 17 — Oscillation of malleus when external canal is at an angle to wave front of shock pulse. 1000 cycle timing wave.

audiogram is found in other kinds of acoustic trauma as for sustained loud noise. For sustained sound the low frequencies are much less traumatizing than the high frequencies even though the low frequencies have a greater masking power than the high frequencies.[8] Therefore one cannot judge the traumatizing power of a noise by its masking effect. One can expose his ears for an hour to a 64 cycle sound so loud that he can just hear a loud shout at the ear and find no depression in the audiogram immediately afterwards. But at the same intensity and duration a 1000 cycle tone produces a marked depression in the threshold, maximum at 1500 to 2000 cycles, that takes three days to disappear. Exposure for an hour to actual industrial noise such as riveting or

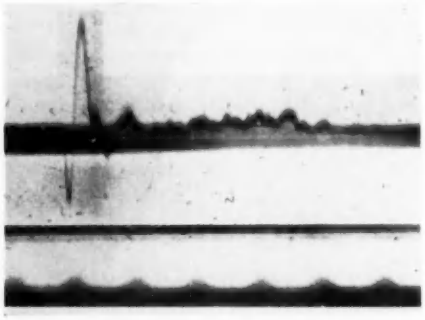

FIG. 18 — Shock pulse response. External canal open. 1000 cycle timing wave.

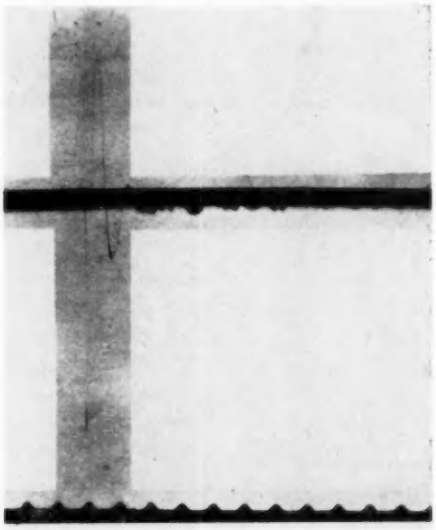

FIG. 20 — Shock pulse response. External canal open. 100 cycle timing wave.

FIG. 19 — Shock pulse response. External canal plugged with beeswax. 1000 cycle timing wave.

* This method could be used in determining susceptibility to acoustic trauma in the armed forces.

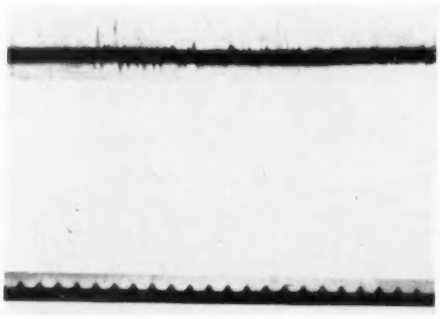

FIG. 21 — Shock pulse response. External canal blocked with glycerine soaked cotton. Note difference in speed of film in figures 20 and 21 as shown in the space between peaks of 1000 cycle timing wave.

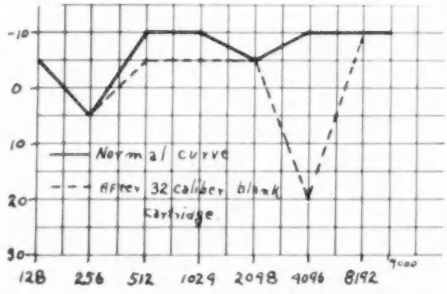

FIG. 22 — Auditory threshold change after exposure to shock pulse of .32 blank cartridge 3 feet from the ear. Subject A = Intermediate frequencies not tested.

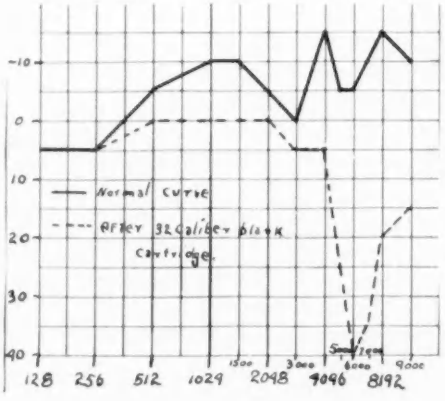

FIG. 23 — Auditory threshold change after exposure to shock pulse of .32 blank cartridge 3 feet from the ear. Subject B = Intermediate frequencies tested.

to noises produced by powerful engines will result in a localized but temporary hearing loss at around 4000 cycles. All the frequencies in the audible range are present under these conditions. Continued exposure produces a permanent depression in the audiogram through this area, spreading to involve the lower frequencies as trauma continues.

The perceptive deafness developed on exposure to the shock pulse is probably due to local injury in the cochlea. The cochlea is so constructed that a shock wave results in a localized brief but powerful reaction at some point along the basilar membrane. Disruption and degenerative changes in the organ of Corti have been reported.[5] Tinnitus may be severe and lasting. But some degree of improvement of tinnitus with recovery of function is regularly observed. The audiogram shows greatest loss when made immediately after exposure. Recovery takes place in a few hours to a few days. After that no further recovery should be expected.

Besides injuring the perceptive mechanism the shock pulse may produce more gross injury to the ear, i.e., rupture of the drum, driving cerumen plugs against the drum or even fracturing the temporal bone. These war injuries have been described by many competent clinical observers, and rules for their care have been formulated.[1,2,3] We have all seen examples of these injuries in civilian practice and I will not go into them because I can add nothing new about their care. For greater realism the results of one such study[4] may be transposed to a street outside this hotel. What I have mentioned about the shock pulse may now help us to understand what would happen to our ears if a bomb exploded nearby.

If a 200 pound bomb exploded in the center of State Street, 85 per cent of the middle ear injuries would be due to the shock pulse itself and only 15 per cent

would be due to bomb fragments. Excluding other injuries, everyone on the street within a radius of 50 feet from the bomb would likely have ruptured ear drums with bleeding from the ear. Outside this radius, hemorrhage into the drum and myringitis may occur. Among those in the stores facing the street, but shielded from the shock pulse by a wall, door or partition, only .5 per cent would have ear injuries.

I have limited my remarks this afternoon primarily to a consideration of the shock pulse and have tried to show why the shock pulse produces an auditory sensation and why it can injure the perceptive and conducting mechanisms of the ear. I hope that this may help you to understand better the ear problems that may arise from noise and explosions in civilian as well as in military practice.

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DISCUSSION

HARRIS P. MOSHER, M.D., Marblehead, Mass.: Doctor Myers asked me to pinch hit for Doctor Bunch, but Doctor Bunch, as many of you know, was one of the very best men we had in the science of acoustics. He was a clear thinker and was able to make one understand what he had to say. His subject carried him into the stratosphere, but, as so often happens with some of the others — I almost said most of the others — who discuss acoustics, one did not have to take oxygen to understand him.

Coming to this paper, frankly, I am not a physicist enough to discuss it. It is very splendidly done. It is a paper for me for reference purposes. Doctor Perlman quoted Doctor Miller. Doctor Miller, some of you will remember, spoke before some of the National societies, perhaps before this one, and brought on a whole laboratory of apparatus to explain his findings.

The only two points that I can discuss are these: The use of ear protectors and the effect of war noises on the ear. Ear protectors will protect, but nobody in the army will use them. If they use anything, they grab a piece of cotton and put it in their ears. You cannot get men either in the Army or in the Navy to use regular ear protectors.

Now about the loss of hearing due to various forms of explosives. War is destructive in all its phases, and of the organs of the body which receive constant damage, the ear stands first.

Doctor Bunch and I (we talked this over) were especially worried about the progressive loss of hearing in aviators. He had the feeling that this was undetected in many cases, and that it was a possible cause of some of the recent commercial crashes, due to the fact that the aviator did not hear the radio beam. That worried me because I had some official connection with the Army at that time. It turned out later that that has been remedied and, as Doctor Perlman said, a good helmet will practically protect the hearing.

The conclusion of Doctor Perlman's paper, in which it is stated what happens in the explosion of a high powered shell, what happens when one is near it and then when one is farther away, is very graphic.

SAMUEL IGLAUER, M.D., Cincinnati, Ohio: I did not understand upon whom these experiments were performed, i.e., on animals, on living subjects or upon cadavers.

H. B. PERLMAN, M.D., Chicago, Ill.: In answer to Doctor Iglauer's question, these records were made by fastening a tiny mirror on the head of the malleus in a fresh temporal bone, and stimulating the conducting mechanism by a shock pulse from a blank cartridge fired near the external meatus of the preparation. A suitable optical system and camera enabled us to record the oscillation of the malleus induced by the shock pulse.

I wish to thank Doctor Mosher for his discussion.

THE CORRELATION OF THE PHENOMENA OF CROSSED AND OBLIQUELY CROSSED CYLINDERS WITH CYLINDER RETINOSCOPY AND CLINICAL REFRACTION

CHARLES HYMES, M.D.
MINNEAPOLIS

IT IS my purpose in presenting this paper to discuss the physical phenomena of crossed and obliquely crossed cylinders and to demonstrate how these phenomena serve both as a basis for cylinder retinoscopy as well as for the technic of ascertaining the strength and axis of an astigmatism by means of the cross-cylinder technic. Contrary to current teachings, these physical laws show that there is no physiological basis for the use of the cross-cylinder technic in the demonstration of the power of the sphere nor of the spherical addition for presbyopia.

The simple cylinder.—A cylinder may be defined as a lens that exerts its power at right angles to its axis. It is, however, of greatest importance to remember that it has refractive power in all meridians except one, and that is its axis. Its power will vary from zero along its axis to its full strength at right angles thereto. To find the power of a cylinder at any meridian, take the number of degrees of this meridian from the axis, multiply it by the power of the lens and divide by 90.

$$F = \frac{\text{No. of degrees} \times \text{power of lens}}{90}$$

F equals power of cylinder at a given meridian. Thus a plus 4.00 cylinder will exert the power of a plus 2.00 cylinder at 45 degrees from its axis.

The crossed cylinders.—Two cylinders of the same sign and of equal strength placed at right angles to each other are equivalent to a sphere of the same strength, while if they are placed with their axes parallel, the result is a cylinder of their combined power. Crossed cylinders of unequal power form a sphere equal to the power of one of the cylinders combined with a cylinder equivalent to the difference between the two. Contrariwise, any sphero-cylinder combination may be resolved into two crossed cylinders.

Thus

(1) $+ 2.00 \text{ cyl.} \times 90 \bigcirc + 3.00 \text{ cyl.} \times 180 = + 2.00 \text{ sph.} \bigcirc + 1.00 \text{ cyl.} \times 180.$

(2) $- 1.00 \text{ sph.} \bigcirc + 2.00 \text{ cyl.} \times 90 = - 1.00 \text{ cyl.} \times 180 \bigcirc + 1.00 \text{ cyl.} \times 90.$

Obliquely crossed cylinders.—Similarly obliquely crossed cylinders have their sphero-cylindrical equivalent. Methods of computing this equivalent have been described by Prentice, Jackson and others. In 1900, Thompson published a trigonometric method and formulae for the transposing of obliquely crossed cylinders into their sphero-cylindrical components.

Presented at the Forty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, October 11-14, 1942, Chicago, Illinois.

From the Department of Ophthalmology, University of Minnesota Medical School.

His formulae are:

1.

$$\text{Cyl.} = \sqrt{P_1^2 + P_2^2 + 2P_1 P_2 \cos. 2A}$$

$$\text{Sphere} = \frac{P_1 + P_2 - C}{2}$$

where C is the resultant cylinder, S is the resultant sphere, P_1 and P_2 are the original oblique cylinders and A is the angle between the oblique cylinders.

The axis of the resultant cylinder is found by means of the following formula:

2.

$$\sin. 2b = \frac{P_2 \sin. 2A}{C}$$

where b is the new angle formed between the resultant cylinder and the first of the obliquely crossed cylinders.

One may get a better understanding of the function of obliquely crossed cylinders by placing two such cylinders before a schematic eye, or better still before an emmetropic eye which is under the influence of a cycloplegic, and proceed to neutralize the resultant sphero-cylindrical reflex by means of retinoscopy. This is the clinical method of transposing obliquely crossed cylinders into their sphero-cylindrical equivalents. It is in fact objective evidence of the behavior of obliquely crossed cylinders.

The following rules will be found to apply to the obliquely crossed cylinders:

(a) If the cylinders are of equal strength and of the same sign, the cylinder of the resultant sphero-cylindrical equivalent will be found midway between the axes of the two component cylinders.

(b) If the cylinders are of unequal power and of the same sign, the resultant axis will be closer to the stronger lens but between the axes of the obliquely placed cylinders.

(c) If the cylinders are of different signs, then the resultant axis will lie outside the angle formed by the oblique cylinders.

The reflexes observed in retinoscopy follow these very same rules, and by employing both spheres and cylinders to neutralize them, one makes use of the laws governing crossed cylinders.

Cylinder retinoscopy. — Cylinder retinoscopy is not generally employed as a technic. Therefore, in order to correlate the laws of crossed and obliquely crossed cylinders with cylinder retinoscopy, some remarks concerning this technic are in order.

Except in cases of muscle anomalies, the precise correction of the sphere is not of great moment; however, the exact correction of the astigmatism is of utmost importance. In cylinder retinoscopy a residual accommodation in eyes under a cycloplegic or mydriatic does not interfere with the determination of the astigmatism because even if the eye does accommodate, the astigmatism is not altered. The patient is asked to look directly into the retinoscopic light. This naturally stimulates his residual accommodation but permits the measurement of the ammetropia at the exact center of the macula. The spherical error is always measured first after which the cylinder is placed before the eye. The power of the cylinder is changed as well as its direction until the cylindrical reflex is entirely neutralized.

We are all familiar with the type of retinoscopic reflex exhibited by hyperopia and hyperopic astigmatism as well as myopia. In myopia the reflexes are more difficult to see and evaluate, and since those of hyperopia are more easily observed, in compound myopic astigmatism one corrects the most myopic meridian first with a minus sphere, thus transposing the astigmatism to the plus variety at the opposite axis. From this point on, the retinoscopy proceeds as in

hyperopic astigmatism. In mixed astigmatism the minus movement is also corrected first for the same reason.

If in cylinder retinoscopy the cylinder is placed in the trial frame at the wrong axis, one observes the phenomena of obliquely crossed cylinders; the cylinder in the trial frame is one, while that of the eye is the second cylinder. The plus cylinder in the trial frame must then be rotated towards the observed "with" movement until the axes of the two cylinders are exactly superimposed. If the correcting cylinder is too weak, a "with" movement appears in the axis of the cylinder in the trial frame. If the correcting cylinder is too strong an "against" movement appears in its axis, while if it is correct, no movement will appear.

At this point it is well to correlate the laws of the simple cylinder and the obliquely crossed cylinders with the phenomena of cylinder retinoscopy. The beam of light must be made to move over the eye at right angles to the principal meridians both in the determination of the sphere and cylinder. This is necessary in order to avoid incorporating into the spherical correction some of the power of the cylinder, for as already has been explained, a cylinder has power in all meridians except in its axis. When the sphere is wrongly determined, it obviously affects the determination of the cylinder.

If, however, the sphere has been overcorrected and the astigmatic reflex properly neutralized, an "against" movement in the sphere is easily observed. The sphere is then reduced in strength and the cylinder correspondingly increased.

The phenomena of obliquely crossed cylinders come into observation when the correcting cylinder is in the wrong axis whether it is correct in strength or not. For the purpose of illustration, it is assumed that the sphere and cylinder are both correct, but the cylinder is in the wrong axis; the reflex of a mixed astigmatism is observed with the retinoscope. Take, for example, an eye with hyperopic astigmatism of two diopters at axis 90. It requires a plus two diopter cylinder at axis 90 for its correction. A plus cylinder obviously must correct a cylinder of the opposite sign. It follows that the eye presents for neutralization a minus two diopter astigmatism.

Sphero — cylindrical equivalent resulting from equal oblique cylinders of different signs.

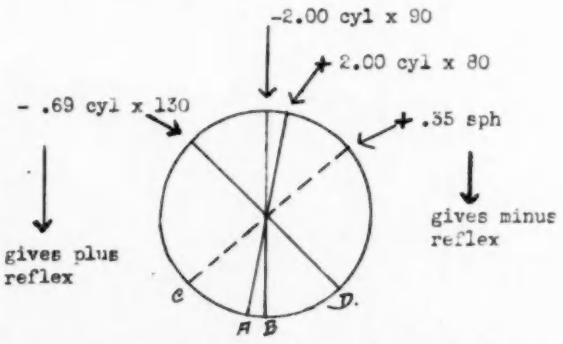


FIG. I (Hymes) — A and B are two equal cylinders of opposite signs. A represents the correcting cylinder (placed in trial frame) and B represents the astigmatism of an eye. (Hyperopic astigmatisms of 2 diopters at 90°.) C and D are the sphero-cylindrical equivalent of A and B. C and D give rise to the reflexes of a mixed astigmatism with the "against" movement at 40° and the "with" movement at 130°.

Now assume the plus two diopter correcting cylinder instead of being placed at 90 degrees is at 80 degrees; the reflexes of a mixed astigmatism of minus .35 sphere together with a plus .70 cylinder axis 130 degrees appears. This is in accordance with the formula of Thompson referred to above. The further away from axis 90 the correcting trial frame cylinder is rotated, the greater the amount of mixed astigmatism that arises, and the further away the plus element of the mixed astigmatic reflex is from the correcting cylinder. Rotating the correcting cylinder towards the 90 axis diminishes the mixed astigmatism, and it disappears entirely when the correcting cylinder is exactly at 90 degrees.

The rule is then, when employing plus cylinders in cylinder retinoscopy, to rotate the trial frame cylinder toward the plus reflex in precisely the same manner as is done when employing the cross cylinder for finding the axis of a cylinder in manifest refraction. The correct axis always lies between the plus axis of the mixed astigmatism and the false position of the trial frame cylinder.

The cross cylinder. — The physical laws of cylinders apply as well to the cross cylinder as it is used in manifest refraction. Here again a knowledge of these principles makes it easier to understand the technic and value of the cross cylinder.

To find the correct axis of the cylinder, place the approximate cylinder in the trial frame at the axis determined by a previous retinoscopy performed either with or without the use of a cycloplegic. The cross cylinder is now placed over the trial frame cylinder which, let it be assumed, is at axis 90 degrees, so that the plus component of the cross cylinder stands alternately at 45 degrees and at 135 degrees. The cross cylinder is twirled at these two axes and the patient is asked in which of the two positions of the cross cylinder the vision is clearest. If vision is equally blurred in both positions, then the trial frame cylinder stands at the correct axis. If the patient states that vision is clearest when the plus component of the cross cylinder is at 45 degrees, the trial frame cylinder must be rotated toward the plus of the cross cylinder, that is, toward the 80 degree axis of the trial frame.

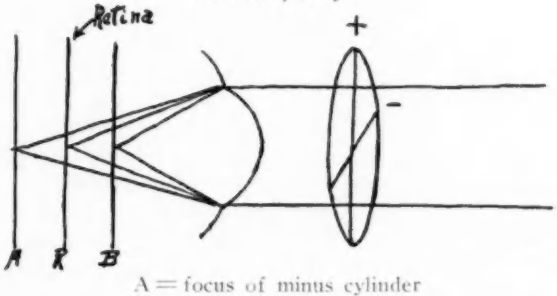
If the true axis of the simple hyperopic astigmia is at 80 degrees and the trial frame cylinder is at 90 degrees, a mixed astigmatism appears with its plus axis at 40 degrees and its minus axis at 130 degrees. Placing the cross cylinder with its plus component at 45 degrees will naturally tend to correct the plus element of the mixed astigmatism at 40 degrees, and simultaneously, the minus component of the crossed cylinder tends to correct the minus element of the mixed astigmatism at 130 degrees. It follows that vision will be much clearer when the plus component of the cross cylinder is at 45 degrees than it would be with the plus of the cross cylinder at 135 degrees.

The trial frame cylinder is rotated toward the side of better vision and the process is repeated until the patient can make no choice. The vision is then equally blurred, and the cylinder in the trial frame is at the correct axis.

To determine whether or not the correcting cylinder is of the proper strength, the cross cylinder is held before the trial frame with its plus and minus axes alternately parallel to the axis of the correcting cylinder. If vision is clearer with one or the other, one simply adds or subtracts from the cylinder in the trial frame; adding when the plus component of the cross cylinder gives clearer vision and subtracting when the minus component gives clearer vision. If the patient states that vision is equally blurred, then the cylinder in the trial frame is correct for power.

It has been taught that the cross cylinder may be used to build the sphere of a refraction as well as to determine the correctness of the spherical addition in presbyopia. At first glance, this would seem to be true because of the action of the spherical component of the cross cylinder. However, to add or to subtract from the sphere, one leaves out of account the action of the cylindrical component of the cross cylinder. It must be remembered that the cross cylinder acts at right angles to both its components. It converges rays in one meridian and diverges them equally at the opposite meridian simultaneously. The patient with the emmetropic eye will respond that the cross cylinder causes equal blurring in all meridians, while the patient with a plus .25 cylinder at 90 degrees states that vision is less blurred when the plus component of the one-fourth diopter cross cylinder is held at 90 degrees than it is when the plus component is at 180. The reason is that the horizontal rays, in such an eye, are focused behind the retina and the plus component of the cross cylinder at the 90 degree axis

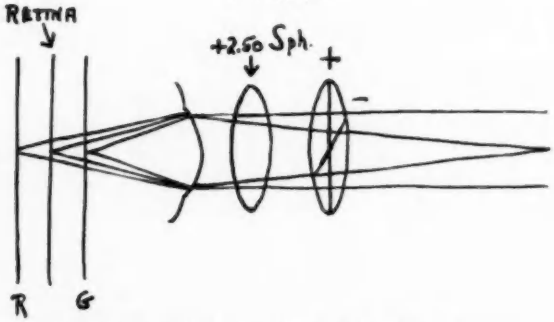
Emmetropic Eye



A = focus of minus cylinder
B = focus of plus cylinder

FIG. 2 (Hymes) — A and B are equidistant to R, the retina. Vision is thus equally blurred in all meridians. If the patient is able to make a choice as to clearness of vision, astigmatism is present.

Presbyopia



R = Minus cylinder — focus behind retina.
G = Plus cylinder — focus in front of retina.
R and G are equidistant to retina.

FIG. 3 (Hymes) — R and G are the foci of the minus and plus components of the cross cylinder placed before the + 2.50 spherical addition to the presbyopic eye. Being equidistant to the retina, they cause equal blurring in all meridians. If patient is able to make a choice as to clearness it indicates the presence of astigmatism rather than the need for more or less spherical addition.

converges them directly upon the retina, while the minus of the cross cylinder diverges the vertical rays behind the retina. Thus only the vertical rays are out of focus, while if the cross cylinder is held with the minus component at the 90 degree axis, the horizontal rays in this eye are diverged still further behind the retina while the vertical rays which formerly focused directly upon the retina are now converged in front of the retina. From this it follows that one may not add or subsract from the sphere as a result of cross-cylinder testing, as the cross-cylinder test is simply one of comparison of diffusion images.

As for its value in presbyopia, the same thing must be true. Here the eye is first made emmetropic for distance and the addition of sphere is made for the desired near point. The cross cylinder can be of no assistance in determining the correctness of the additional sphere for what is true of distant vision must also be true of near vision.

In conclusion it may be said:

1. That there is an exact physical and physiological basis for the technic of the cross-cylinder and cylinder retinoscopy, the very same laws applying to both.
2. That the cross cylinder may be used only to determine the amount and axis of an astigmatism.
3. That in the cross-cylinder technic there is no physiological basis for the determination of the power of the sphere nor of the spherical addition for presbyopia.

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6. Thompson, S. P.: Quoted in Southall test on Mirrors, Prisma and Lenses, 3rd Ed., 1933.

DISCUSSION

W. Howard Morrison, M.D., Omaha, Nebraska: The paper just read by Doctor Hymes contains too much detail in physiologic optics for one to absorb readily. I am certain most of you will benefit by reading and analyzing this contribution after it is printed in the Transactions.

In discussing the simple cylinder Doctor Hymes uses the formula:

$$\text{r} = \frac{\text{No. of Degrees} \times \text{Power of Lens}}{90}$$

This formula according to Cowan[1] is not exactly true. It is, however, easy to remember and use. To be exact it should read as follows:

$Dm = Dc \sin^2 a$

Dm = Power in any chosen meridian

Dc = Total cylinder power

a = Angle between axis meridian and chosen meridian

Doctor Hymes states that as a technic, cylinder retinoscopy is not generally used. I would rather believe that cylinder retinoscopy is employed by most refractionists who use the retinoscope expertly.

I believe some attention must be paid to the sphere in using the cross cylinder to determine the strength of the cylinder. Anyone who uses the cross cylinder finds that the amount of the cylinder cannot be increased indefinitely without decreasing the spherical strength. Conversely the cylinder cannot be greatly reduced without increasing the amount of the sphere. This means that the cross cylinder is not used for determining the sphere alone, as referred to by some refractionists, but it does imply that each time more or less cylinder is indicated by the cross cylinder a sphero-cylinder change must be made.

I again refer to Cowan[2] who states that with a cross cylinder "We are able to add either positive or negative cylindric power and at the same time reduce the spheric power in the same denomination and in the same proportionate strength. This differs from the use of plane cylinders for the same purpose."

When making such sphero-cylinder changes one must understand the means of obtaining a spherical equivalent; that is, the spherical equivalent of a simple cylinder is equal to one-half the power of the cylinder. For example, the spherical equivalent of a plus 4.00 cylinder is a plus 2.00 sphere, or a minus 5.00 cylinder is a minus 2.50 sphere.

The spherical equivalent of a compound is determined by adding one-half of the power of the cylinder to the spherical element of the compound. For example, the spherical equivalent of

$$S + 2.00 \supset C + 4.00 \text{ Ax. } 90 = + 4.00 \text{ sphere}$$
$$S - 2.00 \supset C - 4.00 \text{ Ax. } 180 = - 4.00 \text{ sphere}$$
$$S + 3.75 \supset C - 4.00 \text{ Ax. } 180 = + 1.75 \text{ sphere}$$

The refractionist employing the use of the ¼ D cross cylinder uses his knowledge of the spherical equivalent in this manner: If in a combination of S + 3.00 ⊃ + C + 2.00 Ax. 90, the cross cylinder indicates less cylinder, the next step would be to reduce the cylinder by ¼ diopter, bearing in mind that in reality the sphere has been increased ⅛ diopter. If the cross cylinder again demands that the cylinder be decreased by ¼ diopter, then not only is the cylinder again reduced but the sphere also is altered. The new lens combination then is S + 3.25 ⊃ + 1.50 Ax. 90. One-half of the amount the cylinder has been reduced has been added to the sphere.

Prangen[3] says, "When it is used for strength the cross cylinder alters both the sphere and the cylinder, and this alteration should be visualized. If the cylinder effect alone is thought of and the cylinder is changed without the sphere being checked, an increasingly distorted image will be produced and the interval of Sturm will be exaggerated instead of corrected." Prangen credits J. C. Copeland of the Riggs Optical Company with calling to his attention the rule of spherical and sphero-cylindric equivalents.

I have had no experience with obtaining by means of cross cylinders the spherical addition necessary in presbyopia. I do, however, understand that some men, particularly optometrists, use a grid chart of horizontal and vertical lines placed at the reading distance and by means of a pair of cross cylinders balance the horizontal and vertical lines as to blackness by adding or decreasing spheres in the trial frame. A cross cylinder is placed before each eye with the minus axis at 90; if the patient with presbyopia needs more plus, the vertical lines will be blacker. Less sphere is indicated if the horizontal lines appear blacker.

I have enjoyed hearing this paper and in particular have I profited by the thought provoked by the essayist's remarks.

1. Cowan, Alfred: Refraction of the Eye, Ed. 1, Philadelphia, Lea & Febiger, 1938, p. 250.

2. Cowan, Alfred: Refraction of the Eye, Ed. 1, Philadelphia, Lea & Febiger, 1938, p. 268.

3. Prangen, A. de H.: Some problems and procedures in refraction, Arch. Ophth., 18:432 (Sept.) 1937.

CHARLES HYMES, M.D., Minneapolis, Minn.: There are several points that need to be cleared up in Doctor Morrison's discussion of my paper. He believes that cylinder retinoscopy is employed by most refractionists who use the retinoscope expertly. This does not tell us whether that group comprises a majority of refractionists. Such a recent text as Maxwell's book on refraction states that in cylinder retinoscopy, the placing of the correcting cylinder in the wrong axis creates the reflex of a mixed astigmatism which is practically impossible to correct. Maxwell thus inferentially advises against the use of cylinder retinoscopy as a technic. In the body of my paper this very point is discussed both as to its neutralization by means of shifting the correcting cylinder toward the true axis of the astigmatism of the eye as well as its actual measurement by means of minus sphere and plus cylinder.

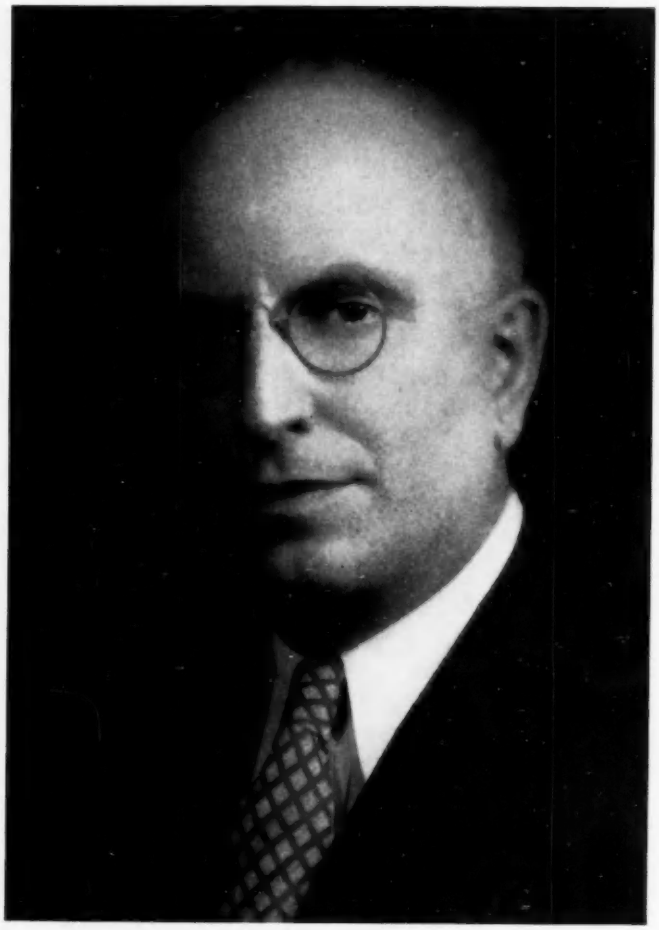
Doctor Morrison apparently confused the clinical sphero-cylinder equivalents (plural) discussed by Prangen with the geometric sphero-cylindrical equivalent of obliquely crossed cylinders which is the basis of my paper. Prangen's sphero-cylinder equivalents are equivalents of a sphere and cylinder and not of obliquely crossed cylinders. In the example cited by him in his very fine article and quoted by Doctor Morrison, he gives 10 different equivalent formulae for the writing of what he refers to as an equivalent prescription. After stating that the full correction of the astigmatism would be ideal, Prangen goes on to say that it is often necessary to give the

eye a compromise image or partial correction for the astigmatism by partially obliterating the interval of Sturm. It is these compromise corrections which he refers to as sphero-cylinder equivalents which should be tried before eyes that reject full correction because previously glasses had not been worn. Any one of several of these compromise formulae may be equally acceptable to the same eye.

Perhaps it was the similarity in terminology that was the confusing element, but it is well to emphasize that there can be but one answer to a geometric problem and not several.

I have but one more suggestion to make regarding the use of the cross cylinder to determine the correctness of the sphere or the spherical addition for presbyopia and that is, cross cylinders refract rays as cylinders and not as sphere and cylinder. Thus the combination of — .50 sph. + 1.00 cyl. axis 90 refracts rays as a cross cylinder, the minus .50 sphere diverges rays at all meridians and the superimposed plus 1.00 cyl. axis 90 converges all horizontal rays to an equal distance in front of the original point of focus. Thus only the vertical rays continue to be diverged by the minus .50 sphere. In other words, the function of the minus sphere has been changed to a minus cylinder. One cannot employ a simple cylinder to test the accuracy of a sphere.

Guest of Honor



RALPH ALBERT FENTON, A.B., M.D., Sc.D.

Graduate of the University of Oregon and Northwestern University Medical School; Trustee of the American Medical Association; Fellow of the American College of Surgeons; Member of the American Board of Otolaryngology; Past President of the Pacific Coast Oto-Ophthalmological Society; Member of the American Academy of Ophthalmology and Otolaryngology, American Laryngological Association (Casselberry Prize with Dr. O. Larsell, 1928; De Roaldes Medal, 1943), American Otological Society, American Laryngological, Rhinological and Otological Society, and of the Collegium Oto-Rhino-Laryngologicum; Professor and Head of the Department of Oto-Laryngology, University of Oregon Medical School; Colonel, Medical Reserve, U. S. Army; Delegate on the part of the U. S. to the Sixth International Military Medical Congress, The Hague, 1931; on active duty December 1917 to August 1919; to France with University of Oregon Base Hospital No. 46, 1918; Attending Surgeon, Army Schools, Langres; Evacuation Hospital No. 1, Toul sector; Consultant in Ophthalmology, Army of Occupation, Coblenz, Germany.

TRANSACTIONS
American Academy of Ophthalmology and Otolaryngology
Official Publication of the American Academy of Ophthalmology and Otolaryngology
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WILLIAM L. BENEDICT, Rochester, Minnesota, *Editor*
W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*
Printed by the Douglas Printing Company, 109 North 18th Street, Omaha, Nebraska

All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, the American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

Committee Activities

Special Survey

The Committee on the Conservation of Hearing has directed Dr. Marvin F. Jones of New York to prepare a treatise on *Surgical Treatment of Impaired Hearing; The Results Obtained by Using the Lempert Endaural Fenestration Technic* for presentation at the meeting of the Academy in Chicago next October. The paper will include experience with the *Fenestration Operation* compiled from the reports of otologists who have done this operation.

It is requested by the Committee that all otologists aid in this survey by forwarding to Doctor Jones immediately answers to these questions:

1. How many fenestration operations have you done?
2. Do you expect to continue using the fenestration technic?
3. How long did you prepare for the first operation on the living?
4. Approximately how many dissections did you do?
5. Under whose supervision did you work?
6. How many patients: (A) Admitted a useful improvement in hearing? (B) Said they were not improved? (C) Claimed to have worse hearing after the operation?
7. What complications have you observed?
8. How many of your patients required reoperation?
9. How long has the improvement in hearing which followed operation been maintained in your most satisfactory case?
10. Have you: (A) Any additional information based on personal experience which you think should be presented to otologists? (B) Any confidential communication which might be of value in directing me toward an accurate opinion.

Please direct your communications to Dr. Marvin F. Jones, 121 East 60th Street, New York, New York.

OFFICIAL NOTICES

FORTY-EIGHTH ANNUAL CONVENTION

American Academy of Ophthalmology and Otolaryngology

OCTOBER 10TH TO 13TH, 1943

Palmer House

Chicago, Illinois

Convention Call

Under the authority of the Constitution and with the approval of the Council, I herewith proclaim that the 48th Annual Convention of the American Academy of Ophthalmology and Otolaryngology will convene at the Palmer House, Chicago, Illinois, on Sunday, October 10th, 1943, and continue through Wednesday, October 13th, 1943.

JAMES A. BABBITT, M.D.
President

Attest:
WILLIAM L. BENEDICT, M.D.
Executive Secretary-Treasurer

Local Committees

WALTER H. THEOBALD, M.D.
General Chairman

Reception Committee

G. Henry Mundt, M. D., Chairman
Thomas D. Allen, M.D.
Howard C. Ballenger, M.D.
I. F. Barnett, M.D.
Beulah Cushman, M.D.
John F. Delph, M.D.
Earl B. Fowler, M.D.
Ira Frank, M.D.
Thomas C. Galloway, M.D.
Glenn J. Greenwood, M.D.
George P. Guibor, M.D.
L. G. Hoffman, M.D.
Paul H. Holinger, M.D.
Arlington C. Krause, M.D.
Vernon M. Leech, M.D.
John R. Lindsay, M.D.
William A. Mann, M.D.
Frank J. Novak, Jr., M.D.
Samuel J. Pearlman, M.D.
Samuel Salinger, M.D.
George E. Shambaugh, Jr., M.D.

Committee on Arrangements

Frank J. Novak, Jr., M.D., Chairman
Carl H. Christoph, M.D.
Richard C. Gamble, M.D.
Elmer W. Hagens, M.D.
George T. Jordan, M.D.
Alfred Lewy, M.D.
Samuel J. Meyer, M.D.
Glenn W. Nethercutt, M.D.
Carl F. Schaub, M.D.
Gail R. Soper, M.D.

Scientific Exhibit Committee

O. E. Van Alyea, M.D., Chairman
Georgiana Dvorak-Theobald, M.D., Consultant

Ladies' Committee

Mrs. Sanford R. Gifford, Chairman
Mrs. Frank J. Novak, Jr., Co-Chairman

Council Meetings

The Council of the American Academy of Ophthalmology and Otolaryngology will convene at 10 o'clock, Sunday morning, October 10, in the Executive Office, Room 403, Palmer House, Chicago, and will meet on Monday, Tuesday and Wednesday mornings of that week from 7:30 to 9 o'clock to transact such business as may come before it. Members desiring to be heard or to present business will confer with the Executive Secretary for appointments.

JAMES A. BABBITT, M.D.
President

Publicity

Contributors to the scientific program are requested to send information regarding their contributions to Dr. Erling W. Hansen, Medical Arts Building, Minneapolis, Minnesota, Secretary for

Public Relations, at the earliest possible moment. Those contributing papers should send a copy of the paper or else a comprehensive abstract. Those contributing to the scientific exhibit should send a description of the exhibit and an explanatory statement clearly indicating the important features of the exhibit.

JAMES A. BABBITT, M.D.
President

Examination by the American Board of Otolaryngology

The next examination by the American Board of Otolaryngology will be held in Chicago, October 7, 8 and 9, 1943. The didactic examination will be given at the Palmer House and the clinical examination at the Illinois Research Hospital.

DEAN M. LIERLE, M.D.
Secretary

Examination by the American Board of Ophthalmology

Part I of the examination (for specially assigned candidates) will be held at the Edgewater Beach Hotel, Chicago, October 7 and 8. This examination will include the following subjects: Anatomy and Embryology, Perimetry, Therapeutics and Operations, Optics and Visual Physiology, Relation of the Eye to General Diseases, Pathology, Bacteriology and Immunology.

The practical and clinical examination, part II, will be held at the Illinois Eye and Ear Infirmary October 9. This will include the following subjects: External Diseases—Slit Lamp; Ophthalmoscopy; Histology — Histopathology; Ocular Motility; Refraction—Retinoscopy; Practical Surgery.

JOHN GREEN, M.D., *Secretray*

Registration

MISS CLARE MCGOVERN, *Registrar*

1. Advance orders for conference tickets will be filled for only members whose 1943 dues have been paid.
2. Receipt for 1943 dues is essential for registration. Members without cards will be required to procure duplicates previous to registration.
3. Instructional conference tickets will be sold to only those registered and with badge.
4. To facilitate registration and the transaction of such other business as is necessary at the Registration Desk, sections will be provided for:

   A. Advance Orders.

   B. Duplicate Receipt Cards.
   Payment of Dues.
   Filing of Academy Applications.
   Registration of Residents exempt from fee (previously arranged for by Head of Department of Institution where training).
   Registration of Academy candidates listed in July-August issue of the TRANSACTIONS and eligible for election at this meeting (exempt from fee).

   C. Non-Members (fee $5.00).
   Candidates whose applications were received too late for listing in July-August issue of the TRANSACTIONS (fee $5.00).

   D. Purchase of Instructional Conference Tickets ($1.50 each period).

   E. Purchase of all dinner tickets (Alumni, Inter-American Relations, etc.).

   F. Purchase of instructional supplies (abstracts, covers, indexes, monographs).
   Orders for Bound Volume of TRANSACTIONS.
   Subscription to bi-monthly issues of TRANSACTIONS.
   Orders for Pathological Atlases —Ophthalmic and Otolaryngic.

   G. Controversies, Miss McGovern.

Main Registration Desk in the Technical Exhibit Hall — Fourth Floor

The Regulation Badge

secured at time of registration is essential for admittance to all activities.

Please Note. — A badge will be furnished at the time of registration upon presentation of receipt card for 1943 dues. Members who fail to bring 1943 receipt card to Chicago will be required to obtain a duplicate previous to registration.

Residents Registering as Guests

To obviate unpleasant misunderstandings where Residents, unknown to the Academy Registrar, register as guests, the Council passed the following resolution:

Men in training in accepted residencies, or those now completing a full academic year (nine months) in basic study, registering as guests during the Academy convention period will be relieved of paying a guest fee only when the Department Head concerned officially requests this favor at least 30 days prior to the opening date of the convention.

New Business

Proposals for new activities often are presented too late during the Academy session to afford time for deliberation. Therefore, new items requiring Council action should be presented in writing to the Executive Secretary early so that the Committee concerned may have an opportunity to study the matter prior to presentation to the Council.

Guests

Physicians are welcome to attend the meeting of the Academy and to participate in all the activities. All guests, however, will be required to register and a fee of five dollars will be charged. Academy candidates whose applications are filed previous to August 1 and whose names are published in the July-August issue of the TRANSACTIONS will be exempt from paying this $5.00 registration fee.

Advance Sale of Instructional Tickets

Members are urged to contract for instructional tickets by advance mail order. Please note instructions for reserving tickets on page 499 and on colored insert. Orders for tickets will be filled for members only whose 1943 dues have been paid.

Ladies' Registration

The wives of members and lady guests are urged to register.

Ladies' Headquarters
Rooms 15 and 16, Club Floor

Dues

A Reminder

Are your 1943 dues paid? Because of the second-class mail regulations, the TRANSACTIONS can be sent only to members in good standing, and orders for conference tickets will be filled only if 1943 dues are paid.

Those who have not paid dues for 1943 are requested to remit to Dr. W. L. Benedict, Secretary - Treasurer, One Hundred First Avenue Building, Rochester, Minnesota.

Constitutional Changes in Membership Status

Honorary Member

RALPH A. FENTON, M.D.
PORTLAND, OREGON

Honor Society

New members to be announced at Inter-American Relations Dinner, Tuesday evening.

Life Members — 1944

Bardenheier, F. G. A.....St. Louis, Missouri
Beach, S. Judd.....Portland, Maine
Blum, Henry N.....New Orleans, La.
Clark, J. Sheldon.....Freeport, Ill.
Collins, J. D.....Northampton, Mass.
Cross, A. E.....Worcester, Mass.
Dow, Frank E.....Northampton, Mass.
Dowling, J. Ivimey.....Albany, N. Y.
Dutrow, H. V. (Major).....U. S. Army
Flatley, T. J.....Moline, Ill.

Grove, W. E.....Milwaukee, Wis.
Hardy, W. F.....St. Louis, Missouri
Inglis, Harry J.....Boston, Mass.
McDowell, N. D.....Rochester, N. Y.
Marshall, Watson.....Pittsburgh, Pa.
Messinger, H. C.....Providence, R. I.
Muncy, William M.....Providence, R. I.
Nutter, C. F.....Nashua, N. H.
Parsons, J. G.....Crookston, Minn.
Ridpath, Robert F.....Philadelphia, Pa.
Smith, George B.....Rome, Georgia
Steim, J. M.....New Kensington, Pa.
Thompson, Peter H.....Boston, Mass.
Turner, H. H.....Pittsburgh, Pa.
Weaver, T. Walker.....Wichita, Kansas
Wise, Ralph C.....Mansfield, Ohio
Wright, J. William.....Indianapolis, Ind.

Senior Members — 1944

Benedict, William L.....Rochester, Minn.
Blackburn, W. J.....Dayton, Ohio
Boucher, R. B.....Vancouver, B. C., Can.
Bribach, Eugene J.....Atchison, Kansas
Burns, S. S.....St. Louis, Missouri
Campbell, Duncan A.....Detroit, Mich.
Campbell, W. K.....Long Branch, N. J.
Cowper, H. W.....Buffalo, N. Y.
Cross, G. H.....Chester, Pa.
Curran, E. J.....Kansas City, Missouri
Davis, William Thornwall.Washington, D. C.
Elles, Norma B.....Houston, Texas
Farmer, A. G.....Dayton, Ohio
Forsythe, S. T.....Cleveland, Ohio
Gifford, S. R.....Chicago, Ill.
Gill, E. G.....Roanoke, Va.
Gordon, J. S.....Milwaukee, Wis.
Harbert, John P.....Bellefontaine, Ohio
Hill, C. E.....Toronto, Ont., Can.
Hurff, J. Wallace.....Newark, N. J.
Jones, F. W.....Alton, Ill.
Krieger, William A.....Poughkeepsie, N. Y.
Linn, J. G.....Pittsburgh, Pa.
Lubman, Max.....New York, N. Y.
McDonald, C. L.....Cleveland, Ohio
Macnie, J. S.....Minneapolis, Minn.
Merrill, H. G.....San Diego, Calif.
Miller, Alice R.....West Palm Beach, Fla.
Moore, George H.....Upper Darby, Pa.
Nelson, Charles F.....Cleveland, Ohio
Novak, Frank J. Jr.....Chicago, Ill.
Patterson, W. E.....Minneapolis, Minn.
Prangen, Avery deH.....Rochester, Minn.
Raydin, B. D.....Evansville, Ind.
Sallander, F. W.....Visalia, Calif.
Schwartz, Frederick O....St. Louis, Missouri
Shapero, I. M.....Rochester, N. Y.
Shemeley, William G. Jr.....Camden, N. J.
Sloan, Henry L.....Charlotte, N. C.
Stimson, George W.....Warren, Ohio
Tangeman, H. F.....Cincinnati, Ohio
Vaughn, E. H.....Tyler, Texas
Waugh, J. M.....Willoughby, Ohio
Wolfe, Otis R.....Marshalltown, Iowa
Zehnder, A. Charles.....Newark, N. J.

THE ANNUAL SCIENTIFIC CONVENTION

Sunday, October 10, 2 p.m.

Joint Session

The Joint Session will be called to order with the opening of the scientific program promptly at 2 p.m. in the Grand Ball Room.

Members are requested to register and to complete other items of business at the Registration Desk early so that the important Joint Session will not be interrupted.

President's Reception

5 P.M.

Red Lacquer Room

President James A. Babbitt and Mrs. Babbitt will appreciate the presence of all members honoring Dr. and Mrs. Ralph A. Fenton and the officers of the Academy.

Committee on Conservation of Hearing

sponsors the following

Scientific Program

8 P.M.

Grand Ball Room

What Can Be Accomplished by Conventional Methods of Treatment. Frederick T. Hill, M.D., Waterville, Maine.

Recent Developments in Non-Surgical Therapy. LeRoy M. Polvogt, M.D., Baltimore, Maryland (by invitation).

Surgical Treatment of Impaired Hearing. The Results Obtained by Using the Lempert Endaural Fenestration

Technic. Marvin F. Jones, M.D., New York, N. Y.

Use of Hearing Aids. Walter Hughson, M.D., Abington, Pa., and Scott N. Reger, Ph.D., Iowa City, Ia. (By Invitation.)

The Right to Hear—Motion Picture.

Committee of Industrial Ophthalmology

sponsors the following

Scientific Program

8 P.M.

Red Lacquer Room

SYMPOSIUM

A Program of Ophthalmic Service for Small Plants. Dr. Harry S. Gradle, Chicago, Illinois.

How Can the Ophthalmologist Be of Greatest Service to Big Business in Wartime. Dr. Clarence Selby, Medical Director of General Motors, and Chairman of the Sub-Committee on Industrial Health Problems of Procurement and Assignment.

The Use of Visual Data as an Aid to Increase Production and Efficiency. Professor Joseph Tiffin, Department of Applied Psychology, Purdue University. (Associate member, Joint Committee of Industrial Ophthalmology.)

A Cross Section of Eye Service in Industry. Mr. C. P. Tolman, Safety Engineer of the National Society for the Prevention of Blindness. (Associate member, Joint Committee of Industrial Ophthalmloogy.)

Treatment of Intra-Ocular Foreign Bodies. To be announced.

American Association of Orthoptic Technicians
Fourth Annual Symposium on Orthoptics

8 P.M. Room No. 17

Training and Duties of an Orthoptic Technician. Walter B. Lancaster, M.D., Boston, Massachusetts.
Discussion opened by Miss Lillian Powell, Chicago.

Essential Requirements for a Good Orthoptic Department. Miss Debora Dicke, Milwaukee, Wis.
Discussion opened by R. Townley Paton, M.D., New York, N. Y.

The Orthoptic Treatment of the Phorias. Murray McCaslin, M.D., Pittsburgh, Pa.
Discussion opened by Mrs. Elsie Laughlin, Iowa City, Ia.

Suppression versus Amblyopia. Miss Marjorie Enos, New York, N. Y.
Discussion opened by Beulah Cushman, M.D., Chicago, Ill.

This meeting is open to members and guests of the Academy and to certified orthoptic technicians.

MONDAY, OCTOBER 11
12:45 P.M.

American Association of Eye, Ear, Nose and Throat Society Secretaries

A Luncheon Meeting

President — W. H. Morrison, M.D.
Omaha, Nebraska
Secretary — Kenneth L. Craft, M.D.
Hume-Mansur Building
Indianapolis, Indiana

Dining Room No. 17

NOTICE TO ALL E. E. N. & T. SOCIETY SECRETARIES

The fifth annual meeting of The American Association of Eye, Ear, Nose and Throat Society Secretaries will be held at the Palmer House, Chicago, at the time of the lunch-

con, Monday noon, October 11, 1943, during the session of the American Academy of Ophthalmology and Otolaryngology. As usual the luncheon will be provided through the courtesy of the Academy. You, as the secretary of an Eye or Ear, Nose and Throat society, are cordially invited and urged to attend this meeting, whether you are a member of this Association or not. If you cannot be present, please urge the president or another officer of your society to represent your group at this luncheon meeting.

The program is in the capable hands of Dr. W. E. Keith, secretary of the Kansas City Society of Ophthalmology and Oto-Laryngology, and Director of District No. 7 of this Association. Dr. Keith will present his thoughts upon certain matters of primary importance to secretaries of E.E.N.T. societies and will then conduct a round-table discussion of these and related subjects. Among the topics to be brought up are:

1. A plan for establishing a central clearing house for scientific articles, case reports, movies, etc., for the benefit of E.E.N.T. societies and the Academy.
2. "To fee or not to fee," the question of the proper financial relationship between the doctor at home and his colleague in Military Service whose former patients are being cared for by the stay-at-home.
3. Post-war status of E.E.N.T. societies. A number of societies have disbanded or are considering such action. Such attitude should be emphatically discouraged. This hinges in with:
4. Part to be played by E.E.N.T. societies in helping combat socialized medicine. Now, more than ever, is the time for militant organization.

Sharpen up your thoughts on these matters and come to this meeting prepared to take an active part in the discussion.

KENNETH L. CRAFT, *Secretary*
1002 Hume-Mansur Building
Indianapolis, Indiana

Alumni Dinners

6:30 P.M.

The Academy is pleased to offer its facilities and to assist in arranging for alumni and other group dinners, Monday evening, October 11, 6:30 p.m. (See special list that follows.)

Tickets may be purchased at the Registration Desk or from a representative of each group.

Buffet Supper

7:00 P.M.

Red Lacquer Room

Members and ladies not otherwise engaged are invited to attend this "No Host" function. Procure tickets at Registration Desk — $1.25. A special surprise program has been prepared. Smoker in Grand Ball Room following dinner. Don't miss it.

TUESDAY, OCTOBER 12
12:30 P.M.

Faculty Luncheon

Private Dining Room No. 14

The President, Dr. James A. Babbitt, and the other officers of the Academy invite all those presenting scientific papers, leaders of instructional conferences, participants in the motion picture sections, scientific exhibitors, members of the Home Study Course faculty and Kellogg fellows in ophthalmology to be their guests at luncheon Tuesday noon at 12:30 in Private Dining Room No. 14.

Business Meeting

5:30 P.M.

Crystal Room

All members are requested to attend.

Inter-American Relations Program and Dinner

7 P.M.

Grand Ball Room

$3.00

Procure tickets at Registration Desk.

WEDNESDAY, OCTOBER 13
12:45 P.M.

Honor Society

The members of the Honor Society will meet at 12:45 p.m. Wednesday for luncheon in Private Dining Room No. 4.

Honor Society headquarters will be in the suite of the President, Dr. E. C. Ellett.

Ladies' Program

Ladies' Committee

Mrs. Sanford R. Gifford, Chairman
Mrs. Frank J. Novak, Jr., Co-Chairman

Ladies are requested to register upon arrival. Information desired will be available in the Ladies' Headquarters, Rooms 15-16, Club Floor.

PROGRAM FOR LADIES

SUNDAY, OCTOBER 10

9 a.m.—Registration
Headquarters Room

5 p.m.—President's Reception
Red Lacquer Room

MONDAY, OCTOBER 11

9 a.m.—Registration
Headquarters Room

7 p.m.—Buffet Supper
Red Lacquer Room

TUESDAY, OCTOBER 12

9 a.m.—Registration
Headquarters Room

7 p.m.—Inter-American Relations Program and Banquet
Grand Ball Room

WEDNESDAY, OCTOBER 13

9 a.m.—Registration.

Alumni Dinners

MONDAY, OCTOBER 11, 6:30 P.M.

$2.50

The facilities of the Academy management are placed at the disposal of alumni and other special groups. Tickets may be secured at the General Registration Desk.

It may be necessary to make some changes in room schedules. Therefore, check bulletin board Monday afternoon.

NOTE: Please use private elevators, Northeast corner lobby floor to Club Building.

Barnhill Students
Private Dining Room No. 4
C. H. McCaskey, M.D., Secretary
Guaranty Building, Indianapolis, Ind.

Dr. Joseph C. Beck's Alumni
Private Dining Room No. 7
J. M. Sutherland, M.D., Secretary
Fisher Building, Detroit, Mich.

Bellevue Hospital Alumni
Private Dining Room No. 5
W. W. Wilkerson, Jr., M.D., Secretary
Bennie-Dillon Bldg., Nashville, Tenn.

Cook County Alumni, Wards 20-21
Private Dining Room No. 15
Carl F. Schaub, M.D., Secretary
31 North State Street, Chicago, Ill.

Wells P. Eagleton Alumni
Private Dining Room No. 3
William H. Hahn, M.D., Secretary
15 Lombardy Street, Newark, N. J.

Gill Memorial Eye, Ear and Throat Hospital
Spring Graduate Group
Private Dining Room No. 6
A. L. Bass, M.D., Secretary
Heyburn Building, Louisville, Ky.

Harper Hospital Alumni
Room No. 795
William S. Gonne, M.D., Secretary
David Whitney Bldg., Detroit, Mich.

Illinois Eye and Ear Infirmary
Private Dining Room No. 17
Roy O. Riser, M.D., Secretary
120 Main Street, Park Ridge, Ill.

Institute of Ophthalmology and Presbyterian Hospital, New York
Private Dining Room No. 11
Charles A. Perera, M.D., Secretary
70 E. 66th Street, New York, N. Y.

Chevalier Jackson Alumni
Private Dining Room No. 16
Paul Holinger, M.D., Secretary
700 North Michigan Ave., Chicago, Ill.

Mayo Clinic, Residents and Ex-Residents
Private Dining Room No. 1
Avery deH. Prangen, M.D., Secretary
Mayo Clinic, Rochester, Minn.

Michigan Joint Alumni Group
Canfield Society — Parker Club
Illinois Room
James H. Maxwell, M.D., Secretary
Canfield Society
First Nat'l Bank Bldg., Ann Arbor, Mich.
Ralph O. Rychener, M.D., Secretary
Parker Club
130 Madison Avenue, Memphis, Tenn.

Harris P. Mosher Alumni
House Surgeons — Course Group
Private Dining No. 14
A. L. Beck, M.D., Secretary
421 Huguenot Street, New Rochelle, N. Y.

New York Eye and Ear Infirmary Alumni
Private Dining Room No. 9
John S. Cregar, M.D., Secretary
150 Harrison Street, East Orange, N. J.

New York Post-Graduate
Medical School and Hospital
Oval Room — Club Building
James W. Smith, M.D., Secretary
1016 Fifth Avenue, New York, N. Y.

Tulane Post-Graduate Alumni
Private Dining Room No. 2
Margaret Alexander, M.D., Secretary
Taylor, Texas

University of Minnesota — Dept. of
Ophthalmology and Otolaryngology
Room No. 788
Frank Knapp, M.D., Secretary
Medical Arts Bldg., Duluth, Minn.

University of Pennsylvania
Ophthalmology
Private Dining Room No. 8
E. Gerard Smith, M.D., Secretary
339 North Duke Street, Lancaster, Pa.

University of Pennsylvania
Otolaryngology
Club Building Lounge
Oram R. Kline, M.D., Secretary
514 Cooper Street, Camden, N. J.

Washington University, St. Louis, Mo.
Ophthalmology and Otolaryngology
Crystal Room
B. Y. Alvis, M.D., Secretary
308 North 6th Street, St. Louis, Mo.

Hotel Reservations for the Academy Convention, October 10-13, Palmer House, Chicago, should be made well in advance. Be sure to get return railway ticket.

SCIENTIFIC EXHIBITS

O. E. Van Alyea, M.D., Chairman
Chicago, Illinois

Georgiana Dvorak-Theobald, M.D., Consultant
Chicago, Illinois

American Medical Association, Council on Physical Therapy, Chicago, Illinois

Exhibitor:
HOWARD A. CARTER, Ph.D.

BY INVITATION

A demonstrational lecture will be given at stated hours on the new method of the Council on Physical Therapy for estimating the percentage of useful hearing loss for medicolegal purposes. An audiometer will be in a booth. The hearing of volunteers will be tested, audiograms made and the percentage loss of hearing calculated according to the Council's method. There will also be a discussion on physical and acoustical terms used in connection with deafness.

An announcement on the blackboard in the booth records the time of each lecture.

Army Medical Museum, Washington, D. C.

Exhibitor:
J. E. ASH, Colonel, M. C., U. S.A.

BY INVITATION

Co-Exhibitor
HELENOR CAMPBELL WILDER

BY INVITATION

The exhibits consist of illustrations of:

Section I — Conditions of unusual interest in the Registry of Ophthalmic Pathology.

Section II — "The Laryngeal Node." This exhibit and an article on the same subject, to be presented at the meeting of the Academy, are based on material in the Registry of Otolaryngic Pathology.

OSCAR V. BATSON, M.D.
University of Pennsylvania
Philadelphia, Pa.

Fractures of the Face

This demonstration consists of a collection of skull specimens showing healed fractures about the nose, maxillae and orbits. These fractures represent untreated or improperly treated cases, and display typical locations and the resulting deformities. From the location and the deformity the proper approach for reduction can be readily adduced.

Biological Photographic Association, Chicago Chapter, Chicago, Illinois

A group of photographs will be exhibited, made by the members of the Association, demonstrating the various types of biological photography in which its members participate. Members will be present each day and all day to help you with suggestions in whatever difficulties you might find yourself in in photographing your patients, specimens, operating room scenes, etc.

Cleveland Clinic
Cleveland, Ohio

Exhibitor:
A. D. RUEDEMANN, M.D.
Visual Field Changes—Region of Chiasm

This exhibit will stress the differential diagnostic points in lesions in the region of the chiasm with special reference to chiasmal arachnoiditis.

Columbia University, College of Physicians and Surgeons, New York, N.Y.

Exhibitor:
A. E. BRALEY, M.D.
Diagnosis and Treatment of Epidemic Keratoconjunctivitis

In this exhibit on epidemic keratoconjunctivitis emphasis will be made on differential diagnosis and results of various forms of therapy. Experimental evidence to support therapy will be given. Technic for isolation of the virus and its use in experimental therapy will be shown.

Committee on Conservation of Hearing
DEAN M. LIERLE, M.D., Chairman

The Discovery and Care of Hearing Deficient School Children

The exhibit presents recent information regarding procedures, standardized technics and needed legislation related to a program recommended by the Committee for the early discovery, medical follow-up and educational adjustment of hearing defective school children. The employment of simplified, uniform technics is emphasized. Recent literature and apparatus will be shown.

Department of Public Welfare
Chicago and Kankakee State Hospitals
Chicago, Illinois

Exhibitor:
PAUL B. SZANTO, M.D.
BY INVITATION
and
University of Illinois College of Medicine
Chicago, Illinois

Co-Exhibitors
HANS BRUNNER, M.D.
and
A. R. HOLLENDER, M.D.

Pathology of the Epipharynx

Specimens (gross) showing development of epipharynx and certain diseases involving this structure.

GEORGE GUIBOR, M.D.
Chicago, Illinois

Ophthalmic Prisms

Characteristics and Uses

This exhibit will be a graphic and photographic demonstration of the optical and physical characteristics of plano-prisms, concavo - convex prisms and sphero-prisms. The desirable and undesirable optical effects of prisms always produce physiologic responses which are influenced by fixation ability, visual acuity, projection, correspondence, reciprocal innervation, fusion, degree of paralysis and severity of contracture.

If these optical characteristics and the resulting physiologic responses are known, prisms can be employed in the diagnosis and in the treatment of motor disturbances without increasing the deviation.

Practical methods of using prisms will be shown and discussed, and mistakes in prescribing prisms will be demonstrated.

A. C. HILDING, M.D.
Duluth, Minnesota

Role of Ciliary Action in Production of Pulmonary Atelectasis and Vacuum in Paranasal Sinuses

1. The mechanical factors involved in the development of postoperative atelectasis.
2. Illustrations from an experiment demonstrating partial vacuum in sinuses.
3. Illustrations from experiments demonstrating the production of reduced pressure in the middle ear.

Jewish Hospital, Brooklyn, New York

Exhibitor:
IRA W. MENSHER, M.D.

The Evolution of the Spectacle

A collection of old spectacles, protective lenses and cases will be presented.

The majority of the various types are over one hundred years old. One pair with the leather frames has been identified as one of the first glasses worn by man, fifteenth century. Some are of silver, most are of a base metal, one is of oxhorn. It will be noted that most of the early spectacles were fashioned by hand. Some of the lorgnettes are of genuine hand-carved tortoise shell. A pair of Franklin or split bifocals is in this collection.

Manhattan General Hospital

Exhibitor:
SAMUEL FOMON, M.D., New York, N. Y.
BY INVITATION

Co-Exhibitors:
ROMEO A. LUONGO, M.D.
Philadelphia, Pa.
MORRIS S. BENDER, M.D.
New York, N. Y.
ALFRED SCHATTNER, M.D.
New York, N. Y.
BY INVITATION

Rhinoplasty

1. Series of 50 diagrams (8″ x 10″) showing stages of typical rhinoplastic operation.
2. Plaster casts demonstrating technic in three dimensions.
3. X-rays of recent and old unreduced nasal fractures.
4. Motion pictures showing role of rhinoplasty in restoration of function.
5. Casts of malar-zygomatic compound fractures and methods used for reduction.
6. Wax casts showing common types of nasal deformities and their relation to disturbed function.

Massachusetts Eye and Ear Infirmary
Boston, Massachusetts

Exhibitor:
GEORGE KELEMEN, M.D.
BY INVITATION

Fractures of the Temporal Bone

This exhibit consists of 90 photomicrograms taken from 21 fatalities due to fracture of the skull involving the temporal bone. The nature of the trauma was that occurring in ordinary life: as a result of fast moving vehicles, falls from various

heights, etc. For comparison, cases of craniotomy for delivery are included and some sections called spontaneous fissures. This exhibit deals exclusively with human material; the pictures demonstrate the variations of temporal bone fractures with their effect upon the different component tissues of the external, middle and internal ear. Most of the serial sections were cut in the horizontal plane with the exception of a small group which were cut vertically. All are stained with hematoxylin-eosin.

National Society for the Prevention of Blindness, Inc., New York, N. Y.

Exhibitors:

ISOBEL JANOWICH

BY INVITATION

and

ELEANOR BROWN MERRILL

BY INVITATION

Industrial Eye Safety and Efficiency in the War Program

This exhibit will present graphically the results of an industrial eyesight protection survey of fifty typical plants. Negligence of such aspects as job analysis, visual examination, periodic eye examination and the use of prescription lenses is pointed out. In addition, the exhibit will include an extensive display of various protective devices for safety against mechanical, chemical, fume and splashing hazards to the eyes.

Temple University
Philadelphia, Pennsylvania

Exhibitor:

CHEVALIER L. JACKSON, M.D.

Co-Exhibitors:

JOHN F. HUBER, M.D.

BY INVITATION

and

CHARLES M. NORRIS, M.D.

BY INVITATION

Contemplated Applied Anatomy of the Bronchi and Lungs, and with a Suggested Nomenclature

The correlated anatomy of the bronchial tree and lungs will be illustrated by simple diagrams, specimens and models. Particular emphasis will be placed on the identification of the orifices of the "segmental" bronchi and their relation to corresponding "bronchopulmonary segments." A simple and practical nomenclature will be presented.

University of Chicago
Chicago, Illinois

Exhibitor:

J. R. LINDSAY, M.D.

Co-Exhibitor:

H. B. PERLMAN, M.D.

(1) Meniere's Disease
(2) Eye Movements

(1) Demonstration by photographs of the pathological entity in the inner ear and of the characteristic auditory findings.

(2) Demonstration by means of recordings of the electrical potentials (retinocorneal) of the various types of normal and pathological eye movements.

University of Colorado, School of Medicine, Denver, Colorado

Exhibitor:

ORA L. HUDDLESTON, MAJOR (MC)

BY INVITATION

Co-Exhibitor:

ADALINE S. BULLEN

BY INVITATION

The important physical, anatomical and physiological principles involved in speech production are illustrated. Two methods of graphic analysis of speech are shown. Speech records of normal and abnormal speech including aphasia, stuttering, hysteria and minor speech defects are presented which were recorded by the graphic method of speech analysis.

University of Illinois, College of Medicine
Department of Ophthalmology
Chicago, Illinois

Exhibitor:

ROY O. RISER, M.D.

Therapeutic Agents in Eye Conditions

A review of accepted and recently introduced medications, including antiseptics, local anesthetics, vasoconstrictors, the sulfonamides, foreign proteins, radiation agents, mydriatics and cycloplegics, miotics and question of the proper pH of eye and nose medications.

University of Illinois, College of Medicine
Department of Otolaryngology
Chicago, Illinois

Exhibitor:
NOAH FOX, M.D.

Therapeutic Agents in Ear, Nose and Throat Conditions

The exhibit will consist of a series of panels, pictures and models, portraying the origin, development and usage of various common drugs and other healing aids valuable to the otorhinolaryngologist. These will include the more common bacteriostatics, antiseptics, anesthetics, pollens, radium and x-ray.

University of Illinois
College of Medicine, Chicago, Illinois

Exhibitor:
MAURICE F. SNITMAN, M.D.

Co-Exhibitor:
IRWIN G. SPIESMAN, M.D.

Excluding surgery of the thyroid gland, operative procedures on the neck belong primarily to the otolaryngologist. Laryngectomy, ligations of internal jugular vein, common and external carotid arteries and lingual arteries, exposure of parapharyngeal abscess, removal of bronchial cysts, pharyngeal diverticulum, etc., require a detailed knowledge of the anatomy of the neck. The procedures described above are intimately related to the viscero-vertebral angle of the neck. Diagrams and enlarged photographs of cross sections of the neck will be used to demonstrate in a simplified manner the anatomy and surgical approach to this important angle.

TECHNICAL EXHIBITORS

Grateful appreciation is expressed to the Technical Exhibitors

Acousticon Division, Dictograph Sales Corp., New York, N. Y.
Booths 7 and 8

The Acousticon exhibit will feature a demonstration of how a person's voice sounds to people with normal hearing and how his voice will sound to people having various stages of hearing loss and deafness patterns that the United States Government National Deafness Survey found to exist throughout the population. There will be distributed at this exhibit a course of training exercises compiled by the Acousticon Research Laboratory for rehabilitating the hearing of people with various hearing loss patterns in accordance with the government survey classification.

American Optical Company, Southbridge, Mass. **Booths 42 to 50**

The American Optical Company of Southbridge, Massachusetts, cordially invites everybody to visit its booth where AO ophthalmological diagnostic equipment and scientific instruments made by the AO Spencer lens division will be shown. Trained instrument men will be glad to discuss and demonstrate this equipment with those interested regardless of the fact that, due to war demands and production, very few instruments will be available for delivery to the professions until after the war.

American Sterilizer Company, Erie, Pa.
Booth 14

THE BARTHOLOMEW SAFETY BED for use in ophthalmology. The patient is placed in the bed and taken to surgery for the operation. When the operation is completed the foot pedal of the bed is depressed and the two sides of the bed are tilted upward to form a "trough" in which the patient may lie gently and comfortably immobilized while being removed from the operating room in the bed. The patient may remain in the bed during the postoperative period.

In this "trough" position the head, shoulders and hips of the patient are comfortably supported at either side, and the tendency to twist and turn is therefore greatly diminished. Futher, in cataract cases, for instance, this bed gives the temporarily blinded patient a hitherto unknown sense of security in that the comforting support of the sloping sides of the bed on the body eliminates any feeling the patient may have of unconsciously creeping and falling out of bed.

A back rest is provided permitting the patient to recline in a partially sitting up posture, either with the mattress flat, or when in the "trough" position, with the sides of the bed and the mattress raised.

The Bartholomew Safety Bed for gently immobilizing the patient will be demonstrated throughout the meeting.

Bausch & Lomb Optical Co., Rochester, N. Y. **Booth 60**

At this wartime meeting of the American Academy of Ophthalmology and Otolaryngology, the Bausch & Lomb exhibit will include modern ophthalmic instruments for refraction and diagnosis.

Every ophthlmologist will be interested in "The Human Eye in Anatomical Transparencies," a new publication produced by Bausch & Lomb. This book is now on the press, but will be available for examination at the Academy meeting. In full color, these anatomical illustrations are amazingly lifelike. They are produced by a new method which gives the reader the effect of having in his hands a translucent three-dimensional model of the eye. Layer by layer, ocular structures are revealed in the order of dissection technics. Authoritative text by Doctors Peter Kronfeld and Stephen L. Polyak accompanies the illustrations.

The delivery of some Bauch & Lomb instruments is limited to the military services, but because of their particular interest to Academy members now vitally concerned with the health of a nation at war, we shall make very effort to have a variety on hand for your inspection.

Bausch & Lomb Affiliated Distributors

Riggs Optical Company
White-Haines Optical Co.
Colonial Optical Company
Southeastern Optical Co.
McIntire, Magee and Brown Co.
Booths 11, 12 and 13

The Bausch & Lomb affiliated companies' display will feature the regular Bausch & Lomb instrument line as well as the newest orthoptic instrument, the Wottring Troposcope.

We will also display many of the war time commodities which our companies have been manufacturing during this past year. These are the various types of prescription goggles used in industry.

Folders, descriptive literature and technical papers on ophthalmic lenses and instruments will be available for free distribution.

Belgard, Inc., Chicago, Ill. Booth 36

Belgard, Inc., will exhibit their line of refracting accessories, with Austin Belgard in charge.

Included in the exhibit will be the firm's "Opticheck," a new wartime product for centering lenses, locating axis and measuring prismatic power. This device is made without the use of critical materials and combines a remarkable degree of efficiency with low cost. Also to be shown are a new illuminated portable Visual Acuity Chart, especially suitable for visiting school nurses, bedside refracting and auxiliary refracting rooms, and a Near Point Visual Acuity Chart with illiterate "E" symbol for quantitative measurement of vision at the near point.

Mr. Belgard extends a cordial invitation to Academy members and visitors to drop in.

Belgard-Spero, Inc., Chicago, Ill.
Booth 15

The House of Vision, Belgard-Spero, Inc., will feature contact lenses and molding equipment at the convention this fall. A representative there will be prepared to answer any and all questions concerning contact lenses. The display will also include other ophthalmic specialties.

Cameron Surgical Specialty Company, Chicago, Ill. Booth 25

Introducing the new Cameron Bronchoscopes, Esophagoscopes and Laryngoscopes. See the new Cameron Binocular Prism Loupe, Flexible Gastroscopes and Cavicamera, Color-Flash Clinical Camera, Mirrolite and other recent developments in electrically lighted diagnostic and surgical instruments for all parts of the body. Of interest are our Radio Frequency Knives, Coagulators and Electrified Snares.

F. A. Davis Co., Philadelphia, Pa.
Booth 18

At booth 18 you may examine the newest books on Ophthalmology and Otolaryngology. Also, if, like many other specialists, you are, because of conditions, being forced into practice outside of your specialty, you will be interested in examining the books referred to in the following letter from a noted specialist.

"The Cyclopedia has been of great aid to me as rapid reference and for detailed information on so many problems now unfamiliar to me, which, because of specialization, I have been away from for a number of years, but now must, because of present circumstances, take care of."

The DeVilbiss Company, Toledo, Ohio
Booth 41

Peace time research and visions of things to come have enabled the DeVilbiss Company to develop from plastic, a new line of home use atomizers, a powder blower and vaporizer. Instruments representative of this new line will be exhibited at booth 41.

All spray parts of the atomizers are made of Saran, a tough, chemically resistant plastic, and the inner, or air tube, is heavily silver plated, making the instruments resistant to all solutions. This simplifies the prescribing of atomizers because one instrument is suitable for any solution.

Mr. L. H. Smock will be in attendance at the DeVilbiss exhibit for demonstrating and explaining the many outstanding features provided by this new line.

Doho Chemical Corporation, New York, Montreal, London Booth 28

The Auralgan exhibit consists of a model of the human auricle four feet high together with a series of 24 three dimensional ear drums, modeled under the supervision of outstanding otologists. Each of these drums depicts a different pathologic condition based upon actual case observation and prepared, insofar as possible, with strict scientific accuracy so as to be highly instructive and interesting to all physicians.

Graybar Electric Co., Inc., New York, N. Y. Booth 61

At the Academy convention in Chicago, October 10-13, will be displayed the latest scientific equipment designed by the Bell Telephone Laboratories, manufactured by the Western Electric Company and distributed through the 87 offices of the Graybar Electric Company to dealers throughout the United States.

Featured at the exhibit will be the new low cost, high efficiency Western Electric Telephone Type Hearing Aid.

Guild of Prescription Opticians of America, Inc., Philadelphia, Pa. Booth 29

THE GUILD OF PRESCRIPTION OPTICIANS OF AMERICA exhibit at the annual convention of the American Academy of Ophthalmology and Otolaryngology, to be held in Chicago on October 10-13 this year, will emphasize in detail what the Guild Optician does for the patients of eye physicians throughout the country; i.e., interpreting accurately prescriptions for glasses; compounding, fitting and servicing glasses. They will renew their pledge of service (1) by making no sacrifice in quality or craftsmanship; (2) making no deviation from the Guild standards of service, and (3) making no unwarranted increase in price.

Keystone View Co., Meadville, Pa. Booth 32

Two new Visual Training Units have been released during the current year. They are the Basic Unit for the development of Stereopsis (BU Series) and the Visual Skills Training Unit (AN Series). A selection of twelve stereographs from the AN Series has been added to the Eye Comfort group. It is designated as the Zeta EC Unit. The Basic Unit may be used in the Tel-EYE-Trainer and in the Major Amblyoscopes.

Lea and Febiger, Philadelphia, Pa. Booth 21

At booth 21 Lea and Febiger will exhibit among their recent publications Ballenger's treatise on The Nose, Throat and Ear; Ballenger's Manual of Otology, Rhinology and Laryngology; Gifford's Ocular Therapeutics; Kovacs' Electrotherapy and Light Therapy; Gray's Anatomy; Speed's Fractures and Dislocations; Dyke and Davidoff's book on Roentgen Treatment of Diseases of the Nervous System; Kraines' Therapy of the Neuroses and Psychoses; Boyd's Text-Book of Pathology; Werner's Endocrinology, and other important publications.

The Macmillan Co., New York, N. Y. Booth 23

The new completely revised tenth edition of Sir John Herbert Parsons' long famous DISEASES OF THE EYE, on display at the Macmillan booth 23, should prove of special interest to members of the American Academy of Ophthalmology and Otolaryngology. Also on hand will be a representative collection of Macmillan's standard medical publications.

Mager & Gougelman, Inc., Chicago, Ill. Booth 35

See the plastic artificial eyes at our booth. Plastics bring a new technic to the making of artificial eyes. These plastic prostheses are especially valuable in cases of socket or globe deformation. They furnish a more faithful reproduction of the sound eye and greater comfort to the wearer. They do not shatter, crack, become rough or discolor.

The Maico Co., Inc., Minneapolis, Minn. Booth 33

The MAICO CO., INC. of Minneapolis will offer to visiting otologists a new publication of great interest covering the field of practical audiometry. In addition there will be a display of the latest developments by MAICO in hearing aids and audiometer test equipment, including a display of test instruments used by the Army and Navy for standard hearing tests. Hearing disability calculators will be given to all visiting otologists, gratis. Any otologist who so desires may have a pair of individually molded plastic ear stopples molded for his own ears as "noise eliminators."

Medical Film Guild, New York, N. Y. Booth 66

The Medical Film Guild will emphasize its talking papers in this year's program of "MEDICAL FILMS THAT TEACH." Hospital and Medical Society program chairmen, now faced with depleted staffs because of the war emergency, who desire educational material for their meetings, find that Medical Film Guild's motion picture film text books lessen that important problem. Through grants for post-

graduate instruction, these films, including projection service, are available, without charge to any hospital or medical society meeting and to the medical services connected with the Armed Forces of the United States. The following films will be shown in the booth of the Guild:

Inguinal Hernioplasty — Symposium on Suturing Technic
Asphyxia Neonatorum
Non-Operative Treatment of Paranasal Sinusitis
Otitis Media in Pediatrics
A Clinic on Acute Mastoiditis
Otoscopy in the Inflammations
A Clinic on Petrositis with Meningitis
A Clinic on Sigmoid Sinus Thrombosis
Pharmacology of Respiratory Stimulants
Sutures Since Lister

The C. V. Mosby Co., St. Louis, Missouri Booth 22

A cordial invitation is extended to all physicians attending this year's meeting of the American Academy of Ophthalmology and Otolaryngology to visit booth 22, where the C. V. Mosby Company will display a complete line of medical publications. Of particular interest will be the new Hughes "Reconstructive Surgery of the Eyelids."

V. Mueller and Co., Chicago, Ill. Booths 51 to 57

In addition to the regular line of instruments for eye, ear, nose and throat surgery, V. Mueller and Company (Chicago) will present several new developments of particular interest to ophthalmologists. There will be a large selection of fine eye forceps, hand made, in stainless steel, and a complete display of Mueller-made eye knives, keratomes, etc. The exhibit will also include the latest models of Mueller anesthesia and suction-pressure equipment.

New Jersey Optical Co., Irvington, N. J. Booth 4

A cordial invitation is extended those attending the convention by New Jersey Optical Company to visit booth 4.

Obrig Laboratories, Inc., New York, N. Y. Booth 16

Contact lenses are being used more and more to rehabilitate persons who have had corneal injuries. After the war there will be a greater need for these devices to help restore vision to men who have received corneal wounds.

The Obrig Laboratories will exhibit contact lenses, gonioscopic lenses, microscopy lenses, tinted lenses and the materials for making impressions of the eyes, as well as the equipment for adjusting the fitting of contact lenses.

Many leading ophthalmologists consider contact lenses to be "the glasses of the future."

The all-plastic unbreakable contact lens is 60 per cent lighter than the glass lens; it is inert and easily adjustable and can be ground and polished to any correction.

The new molding material for making impressions of the eyes will be on display. This impression material surpasses anything which has been used to date.

Philip Morris and Co., Ltd., New York, N. Y. Booth 59

Philip Morris & Company will demonstrate the method by which it was found that Philip Morris Cigarettes, in which diethylene glycol is used as the hygroscopic agent, are less irritating than other cigarettes. Their representative will be happy to discuss researches on this subject and problems on the physiological effects of smoking.

George P. Pilling and Son Co., Philadelphia, Pa. Booths 1, 2 and 3

We are devoting almost all our manufacturing facilities to produce urgently needed surgical instruments for the Army and Navy, and therefore we will not be able this year to show a complete line of our specialties as in the past. There will be prominent gaps in the line.

However, extra special efforts are being made to assemble an interesting exhibit, and we feel confident it will be worth your inspection as it always has been in the past.

We hope our friends, therefore, will visit us and as usual every possible effort will be made to serve them.

R. J. Reynolds Tobacco Co., New York, N. Y. Booths 30 and 31

CAMEL Cigarettes will exhibit large detailed photographs of equipment used in comparative tests of the five largest-selling brands of cigarettes. Dramatic visualization of nicotine absorption in the human respiratory tract from cigarette smoke will be demonstrated. Internation-

al news with the CAMEL Cigarette Trans-Lux "Flash Bulletins" may be seen while enjoying a supply of slow-burning CAMEL Cigarettes.

Ritter Company, Inc., Rochester, N. Y. Booths 64 and 65

The Ritter Ear, Nose and Throat Unit has all water, air and electrically operated instruments, all medical bottles and sprays within arms' reach. The Ritter Motor Chair brings greater operating efficiency to you as well as increased comfort to the patient. The Ritter X-ray with its shock proof, easily adjusted head for rapid and accurate radiograph and the Ritter Hydromatic Sterilizer with its automatic and labor saving features are of particular interest to the busy doctor.

Shuron Optical Company, Inc., Geneva, N. Y. Booth 40

The Shuron Optical Company is at war. It is making a great variety of direct war products for the Army, Navy and Lend-Lease. Among these are included mobile optical prescription shops, gas mask frames and cases, Porro Prisms, parts for aircraft cameras, machine guns, parachutes, stereoscopic trainers, Diesel engines, etc.

The Shuron representatives at the Academy meeting will be glad to give more information about the war work and to explain why it is more and more difficult to supply the ever-increasing civilian demand for Shuron frames, mountings and lenses.

Singer Sewing Machine Company, Surgical Stitching Instrument Division, New York, N. Y. Booths 38 and 39

Specially trained demonstrators will exhibit the new Singer Surgical Stitching Instrument. This instrument, which was developed in close cooperation with the surgical profession, has attracted much attention in several recent medical conventions, and clinical trials have definitely proved its success and practicability.

All doctors, nurses and hospital staff members are cordially invited to see this unique contribution to surgery. Motion pictures of operations showing the instrument in use will be shown, and free literature describing the instrument in detail will be available.

S.M.A. Corporation, Chicago, Ill. Booth 24

You are cordially invited to visit the S.M.A. exhibit at booth 24 where clinical deficiency diseases affecting the eye will be illustrated by life-size wax moulages. SMACO nutritional biochemicals of interest to the ophthalmologists will be featured.

Smith, Kline and French Laboratories, Philadelphia, Pa. Booth 37

Come to booth 37 for information on how the physician may save himself time and trouble and spare the patient prolonged visual disability by using the "Paredrine" technic in refractions. Also, we will gladly explain the local use — both medical and surgical —of "Micraform" sulfathiazole in rhinologic and otologic practice. "Micraform" preparations include "Paredrine," Sulfathiazole Suspension, "Micraform," Sulfathiazole Suspension, 5 per cent, and "Micraform," Sulfathiazole Suspension, 20 per cent.

Soft-Lite Lens Co., Inc., New York, N. Y. Booths 9 and 10

Ophthalmologists in increasing numbers find that the neutral absorption afforded by Soft-Lite Lenses gives patients greater comfort when their refraction indicates the need of protection from natural or artificial over-brightness. Soft-Lite Lenses are available in five densities in the finest single vision and bifocal forms — all made by Bausch and Lomb exclusively for the Soft-Lite Lens Company, Inc., New York, Toronto, London.

Frederick Stearns and Co., Detroit, Mich. Booths 26 and 27

Doctors are cordially invited to visit our attractive convention booth to view and discuss outstanding contributions to medical science developed in the Scientific Laboratories of Frederick Stearns and Company.

Our professional representatives will be pleased to supply all possible information on the use of such outstanding products as Neo-Synephrine Hydrochloride for intranasal and ophthalmologic use, Amino Acids for parenteral protein feeding, Mucilose for bulk and lubrication, Fergon (Ferrous Gluconate), Potassium Gluconate, Gastric Mucin, Susto, Trimax, Appella Apple Powder, Nebulator with Nebulin A, and our complete line of vitamin products.

Storz Instrument Co., St. Louis, Missouri
Booths 5 and 6

The Storz Instrument Company is happy to report that again they can take care more promptly of orders and repairs sent in by their civilian customers now that the most urgent work for the Government is completed. For the same reason it is possible to show again a more complete selection of the instruments made in the Storz shop. As always, fine workmanship is being stressed.

Uhlemann Optical Company, Chicago, Ill.
Booths 62 and 63

Exhibit — PHYSICIAN'S QUALITY GLASSES. First to serve the ophthalmologist exclusively. Let us tell you of the service which we have to offer.

The Univis Lens Co., Dayton, Ohio
Booth 58

The Univis Lens Company is introducing no new item this year, but will have on display a rather interesting exhibit of never-marketed lens designs.

Most of the items on display are lenses developed in experimental work by Univis technicians over the years, although some of the multifocals were made at the suggestion and according to the design of various refractionists.

The Williams and Wilkins Co., Baltimore, Maryland **Booth 20**

The Williams and Wilkins Company will have **new editions** of Best & Taylor's "Physiological Basis of Medical Practice," and Armstrong's "Aviation Medicine." Also see the new "Atlas of Anatomy" by Dr. J. C. B. Grant, the first American atlas of anatomy in color. The new "Industrial Medicine," edited by Dr. Wampler, is a timely contribution.

White Laboratories, Inc., Newark, N. J.
Booth 34

Within recent years tremendous advances in vitamin research have added a wealth of clinical data to our knowledge of nutrition.

The intense interest of the laity in the vitamins—often, unfortunately, confused and misled by unauthoritative lay advertising and uninformed "information" — can be properly controlled by the physician's interpretation of the actual usefulness of the vitamins to his patients.

In booth 34 White Laboratories, Inc., presents its complete line of ethically promoted, clinically reputable vitamin preparations. Qualified representatives will be in attendance to discuss the use of White's products in vitamin prophylaxis and therapy. Descriptive literature is available for review, and a cordial welcome is extended.

PROGRAM IN BRIEF

1943 Meeting — Chicago

Pre-Convention Activities

WEDNESDAY, OCTOBER 6

"Get Together Party"

8:30 p.m.—Officers and their wives and members of Local Committees and their wives — Palmer House — Room No. 18

THURSDAY, OCTOBER 7

American Board of Otolaryngology

1:30 p.m.—Didactic Examination — Palmer House

American Board of Ophthalmology

12:30 p.m.—Part I — Examination (for specially assigned candidates) — Edgewater Beach Hotel

FRIDAY, OCTOBER 8

American Board of Otolaryngology

8:30 a.m.—Clinical Examination—Illinois Research Hospital

2 p.m.—Clinical Examination — Illinois Research Hospital

American Board of Ophthalmology

8:30 a.m.—Part I — Examination — Edgewater Beach Hotel

SATURDAY, OCTOBER 9

American Board of Otolaryngology

8 a.m.—Didactic Examination — Palmer House

American Board of Ophthalmology

8:30 a.m.—Part II — Examination — Illinois Eye and Ear Infirmary

Convention Activities

OCTOBER 10 TO 13, 1943

SUNDAY, OCTOBER 10

8:30 a.m.—Meeting of Committee on Inter-American Relations in Otolaryngology (or Pan-American Congress of Otolaryngology) — Private Dining Room No. 8

8:30 a.m.—Meeting of General Executive Committee on Conservation of Hearing — Crystal Room

9 a.m.—Official Opening of Exhibits — Technical and Scientific

9 a.m.—Registration — Exhibit Hall, Fourth Floor

9 a.m.—Ladies' Registration — Rooms 15 and 16 — Club Floor

10 a.m.—Council Meeting — Room 403

2 p.m.—Scientific Section — Joint Session — Grand Ball Room

5 p.m.—President's Reception — Red Lacquer Room

8 p.m.—Scientific Program — Conservation of Hearing — Grand Ball Room

8 p.m.—Scientific Program — Industrial Ophthalmology — Red Lacquer Room

8 p.m.—Scientific Program — American Association of Orthoptic Technicians — Room No. 17

MONDAY, OCTOBER 11

7:30 a.m.—Council Breakfast Meeting — Executive Office — Room 403

7:30 a.m.—Breakfast Meeting—Directors and Officers, American Association of E. E. N. & T. Society Secretaries.

8 a.m.—Registration — Fourth Floor

8:30 a.m.—Instructional Conferences — Seventh Floor

9 a.m.—Ladies' Registration — Rooms 15 and 16 — Club Floor

10 a.m.—Instructional Conferences — Seventh Floor

11:30 a.m.—Instructional Conferences — Seventh Floor

12:45 p.m.—American Association of E. E. N. and T. Society Secretaries — Luncheon—Private Dining Room No. 17

2 p.m.—Section on Ophthalmology — Red Lacquer Room

2 p.m.—Motion Pictures — Otolaryngology—Grand Ball Room

6:30 p.m.—Alumni Dinners

7 p.m.—Buffet Supper — Ladies invited to participate in both dinner and program following — Red Lacquer Room

TUESDAY, OCTOBER 12

7:30 a.m.—Council Breakfast Meeting — Executive Office — Room 403

8:30 a.m.—Instructional Conferences — Seventh Floor

10 a.m.—Instructional Conferences — Seventh Floor

11:30 a.m.—Instructional Conferences — Seventh Floor

12:30 p.m.—Faculty Luncheon — Private Dining Room No. 14

2 p.m.—Section on Otolaryngology — Red Lacquer Room

2 p.m.—Motion Pictures — Ophthalmology—Grand Ball Room

5:30 p.m.—Business Meeting — Crystal Room

7 p.m.—Inter-American Relations Dinner — Grand Ball Room

WEDNESDAY, OCTOBER 13

7:30 a.m.—Council Breakfast Meeting — Executive Office — Room 403

8:30 a.m.—Instructional Conferences — Seventh Floor

10 a.m.—Instructional Conferences — Seventh Floor

11:30 a.m.—Instructional Conferences — Seventh Floor

12:45 p.m.—Honor Society Luncheon — Private Dining Room No. 4

2 p.m.—Section on Ophthalmology — Red Lacquer Room

2 p.m.—Section on Otolaryngology — Grand Ball Room

SCIENTIFIC SECTIONS

Sunday, October 10, 2 p.m.

Joint Session

GRAND BALL ROOM

Presiding Officer:
President, JAMES A. BABBITT, M.D.

SECRETARIES IN CHARGE
Ophthalmology:
Algernon B. Reese, M.D.
Otolaryngology:
O. E. VAN ALYEA, M.D.

Call to Order.....The President
James A. Babbitt, M.D., Philadelphia, Pa.

Announcements. The General Chairman
Walter H. Theobald, M.D., Chicago, Ill.

Introduction of the Guest of Honor
Ralph A. Fenton, M.D., Portland, Ore.

Introduction of Special Guests

President's Address
James A. Babbitt, M.D., Philadelphia, Pa.

Address of the Guest of Honor
Ralph A. Fenton, M.D., Portland, Ore.
The Unity of Medical Science in the Americas

SYMPOSIUM: *Seeing, Hearing, Thinking and Doing—A Problem of American Youth*

Joseph E. Raycroft, M.D., Princeton, New Jersey BY INVITATION
A. D. Ruedemann, M.D., Cleveland, Ohio
A. C. Furstenberg, M.D., Ann Arbor, Mich.

Monday, October 11, 2 p.m.

Ophthalmology — Scientific Papers — Red Lacquer Room
Presiding Officer: Sanford R. Gifford, M.D.
Secretary: Algernon B. Reese, M.D.
Otolaryngology — Motion Pictures — Grand Ball Room
Presiding Officer: Wesley C. Bowers, M.D.
Secretary: O. E. Van Alyea, M.D.

Tuesday, October 12, 2 p.m.

Otolaryngology — Scientific Papers — Red Lacquer Room
Presiding Officer: President, James A. Babbitt, M.D.
Secretary: O. E. Van Alyea, M.D.
Ophthalmology — Motion Pictures — Grand Ball Room
Presiding Officer: William A. Krieger, M.D.
Secretary: Algernon B. Reese, M.D.

Wednesday, October 13, 2 p.m.

Otolaryngology — Scientific Papers — Grand Ball Room
Presiding Officer: President, James A. Babbitt, M.D.
Secretary: O. E. Van Alyea, M.D.
Ophthalmology — Scientific Papers — Red Lacquer Room
Presiding Officer: Sanford R. Gifford, M. D.
Secretary: Algernon B. Reese, M.D.

Authors of Papers

Please be prepared to submit papers to the recorder immediately following presentation.

There will be discussion of papers presented at the scientific sections Monday, Tuesday and Wednesday.

Section on Ophthalmology

MONDAY, OCTOBER 11, 1943

2 p.m.

RED LACQUER ROOM

Presiding Officer:
SANFORD R. GIFFORD, M.D.
First Vice President

Secretary:
ALGERNON B. REESE, M.D.

Address of the First Vice President
SANFORD R. GIFFORD, M.D.
Chicago, Illinois
The Factor of Angiospasm in Ocular Diseases

Epidemic Keratoconjunctivitis Results of Therapy

ALSON E. BRALEY, M.D.
New York, N. Y.

Discusser: HARRY S. GRADLE, M.D.
Chicago, Illinois

Fuchs 53 years ago described 38 cases of a disease that resembled epidemic keratoconjunctivitis. Herbert, in 1900, reported 26 cases in Bombay. Kirwin in 1930 and Wright in 1930 reported extensive epidemics in India. Outbreaks occurred in India and China from 1933 to 1936, in Germany from 1938 to 1940, in Malaya from 1935 to 1938 and in Tasmania in 1941. Holmes in 1941 reported 10,000 cases in Hawaii. Hobson first reported its occurrence in the United States. Cases began appearing in San Francisco in 1941. Hogan and Crawford and de Roetth reported cases from the west coast.

Bacterial cultures from the disease may show bacteria on the conjunctiva. Inoculation of conjunctiva scrapings in baboons are negative. Inoculation of rabbits gives a transient conjunctivitis. Intracerebral inoculation of mice gives a transient encephalitis in some instances. A virus has been isolated by Sanders in mice after first passing inoculum through tissue culture.

The clinical features of the disease consist of edema, lymphoid hyperplasia, enlarged preauricular lymph node, marked tearing with no purulent discharge and typical subepithelial corneal infiltrations. The disease must be differentiated from ocular disease associated with edema, preauricular lymph node involvement, follicular hyperplasia, acute conjunctivitides with a preauricular node, follicular conjunctivitis and allergic conjunctivitis.

The laboratory diagnosis during the acute phase of the disease is difficult. Scrapings of the conjunctiva show predominantly lymphocytes, large mononuclears and very few polys. In the late stages, a laboratory diagnosis is possible by testing for the presence of virus neutralizing substances in the patient's serum.

To date 296 cases have been treated. Of these 209 were treated symptomatically with no effect on the course of the disease. Corneal opacities developed in all of the cases. Ten cases were treated with local application of sulfathiazole sesquihydrate, with little or no change in the clinical course. Twenty-five cases were treated with five per cent sulfathiazole ointment, and in most instances it seemed to increase the symptoms. Three cases were treated with local penicillin with no effect. Ten cases treated with tyrothricin showed some clinical improvement, but all patients developed corneal opacities. Of 35 cases treated with convalescent plasma, the course of the disease was shortened and few or no corneal opacities were present in 32 cases, while in three cases the course of the disease was not shortened, and marked corneal opacities developed. In four cases treated with normal plasma, all followed the normal course of the disease.

The determination by Sanders and Alexander of neutralizing substances in the patient's serum was used as a final test for the disease. In order to evaluate the test and to determine the effect of convalescent plasma on other diseases, patients with Beal's conjunctivitis, severe staphylococcal conjunctivitis, pneumococcal conjunctivitis and severe conjunctivitis of unknown etiology were given convalescent plasma, and after recovery from their primary disease, the test for serum antibodies was made. In all instances no neutralizing substances against the virus could be demonstrated, while in cases with epidemic conjunctivitis treated with convalescent plasma, all showed high antibody titer.

A summary of control methods and treatment of epidemic keratoconjunctivitis will be made.

Chronic Sinusitis as a Possible Etiologic Factor in Some Cases of Chronic Simple Glaucoma

CONRAD BERENS, M.D., New York, N.Y.
and
EDITH L. NILSON, New York, N. Y.
BY INVITATION

Discusser: ALAN C. WOODS, M.D.
Baltimore, Md.

The literature has been reviewed for theories relating to the etiology of chronic simple glaucoma, but most of the causes suggested appear to be secondary rather than primary in character. Focal infection has occasionally been mentioned as a possible etiologic factor, and careful continued observation in some of our cases suggested the nasal accessory sinuses as probable foci.

A study of the case histories of patients with glaucoma under our care reveals that, in a large proportion, the nasal sinuses showed pathology. This was determined by clinical examination and either bacteriologic studies or roentgenograms or both. In certain cases, eradication or improvement of the sinus disease seemed to simplify the control of ocular tension. Cases will be reported in which the association between sinus pathology and glaucoma seemed apparent to us. The association between sinus disease and glaucoma seemed particularly suggestive in cases of unilateral simple glaucoma, in which the glaucomatous eye was usually to be found on the side of the more evident sinus involvement.

Whether the sinus infection is only a secondary factor, superimposed upon an eye anatomically predisposed to glaucoma, or whether it is a primary cause, naturally has not been determined. These studies, however, suggest that chronic sinusitis merits more attention as a possible etiologic factor in chronic simple glaucoma than it has heretofore been given.

The Dickey Operation for Ptosis — Results in 21 Patients and 30 Lids

FREDERICK C. CORDES, M.D.
San Francisco, California
and
ULRICH FRITSCHI, M.D.
San Francisco, California
BY INVITATION

Discusser: SANFORD R. GIFFORD, M.D.
Chicago, Illinois

In 1935 Dickey conceived the idea of correcting ptosis in those cases with normal function of the superior rectus muscle by using a sling of fascia lata through the middle third of that muscle and attaching it to the tarsus. Since that time the Dickey operation has been used at the University of California, and during this period 30 lids have been operated upon on 21 patients with satisfactory results.

It is felt the operation has certain advantages over other procedures. It can be used in small children, in one instance in a 13 months old child. Due to the two point suspension there is no tendency for the inverted V formation of the lid, and the skin folds show the normal conformation. The operation permits adjusting the position of the lid to conform with that of the other lid.

In the series presented, three lids previously unsuccessfully operated upon by other methods were corrected without great difficulty.

The operation has been found to be satisfactory in bilateral complete ptosis, unilateral complete ptosis and partial ptosis in those cases where the action of the superior rectus is normal.

The details of technic of the operation will be presented.

The Value of an Orthoptic Clinic in Private Practice

EDWARD C. ELLETT, M.D.
Memphis, Tennessee
RALPH O. RYCHENER, M.D.
Memphis, Tennessee
and
JEAN S. ROBINSON, A.B.
Memphis, Tennessee
BY INVITATION

Discusser: F. BRUCE FRALICK, M.D.
Ann Arbor, Michigan

The Memphis Orthoptic Clinic has been in operation for 10 years. Organized as a private clinic, open to the patients of any ophthalmologist practicing in Memphis, viewed at first with great skepticism by all of us, it has apparently justified in great degree its cost and maintenance.

A critical review of the patients seen there for diagnosis and treatment indicates that only a small percentage are suitable or available for orthoptic training in that clinic. Of those treated, only two cases of accommodative squint were cured by orthoptic training alone. The clinic was an invaluable aid in diagnosis especially with youngsters under six, and in the treatment of preoperative and postoperative accommodative convergent squint through the correction of amblyopia and false projection. Divergence excess responded well to exercises, and where cooperation was given could be well controlled, but showed a tendency to relapse when the patient was removed from supervised training.

The clinic has justified its expense in the field of the convergence insufficiencies alone. These groups may be designated as a latent convergence insufficiency, where the usual muscle measurements are normal, but the prism divergence-convergence relationship or reading ratio as evidenced on the metronoscope is poor, and a true convergence insufficiency is denoted by definite deficiency of conver-

gence or amplitude of convergence as measured on the phoropter. The former group has been largely overlooked, but includes patients who have difficulty in near work, regardless of normal vision, proper correction of refraction, normal conjunctivae and lacrimal apparatus and normal muscle measurements on the phoropter. Also included are those who find difficulty in becoming adjusted to their first bifocals. All these patients can be relieved of symptoms and restored to work by an increased power in prism convergence induced by exercises under supervision of the orthoptic technician. True convergence insufficiency is the ideal field for orthoptic training.

The orthoptic clinic, although seldom reaching the goal suggested by the enthusiastic apparatus salesman, i.e., the cure of squint by exercises alone, has nevertheless justified its operation by removing from the busy practitioner's office the laborious orthoptic training of patients for which he has neither time nor space, and by aiding him in restoring to normal comfort and appearance many patients with heterophorias and heterotrophias.

Section on Otolaryngology

TUESDAY, OCTOBER 12, 1943

2 p.m.

RED LACQUER ROOM

Presiding Officer:
PRESIDENT, JAMES A. BABBITT, M.D.

Secretary:
O. E. VAN ALYEA, M.D.

Bartholomaeus Eustachius
His Contribution to the Anatomy of the Ear, Larynx and Bronchi

CHARLES J. IMPERATORI, M.D.
New York, N. Y.

Eustachius was an anatomist of superior ability. Excepting for the seven illustrations in his Opuscula that were printed the year of his death, his great contribution to anatomy was not developed until nearly a century and a half afterwards. In this respect, he parallels Leonardo da Vinci, whose work was not rediscovered for over 400 years. Nevertheless, the Eustachian Tabulae were in considerable vogue in medical circles from 1714 to 1800.

The accuracy of most of the illustrations in the Tabulae are such that they might be used, even today.

It is unfortunate that these Tabulae were lost at a time when they would have been so valuable to the medical student. The evidence of their value is manifest for after 140 years, they were recovered and released, and they were used continuously for over eighty years in the teaching of anatomy.

Controversies of those days were bitter, and should they invoke one with the Church, retribution quickly followed. These Tabulae may have been suppressed.

Eustachius was in the midst of the Vesalian discussion, and while he did not repudiate Galen, his accurate Tabulae Anatomicae definitely did so.

With but few exceptions, the brilliant men of that day rarely reached 50 years of life. Eustachius died when he was 54 years while on a journey from Rome. His name attached to the tuba auditiva is just one of the mysteries that occur in anatomical nomenclature and as advanced by Portal, "because of his researches and knowledge of its anatomy, he was not embarrassed to attach his name to it." This statement implies that Eustachius was cognizant of this.

Regarding his knowledge of the function and physiology of the tube, very little can be said. A very careful translation of his Opuscula may reveal some further facts in this regard. In any event, one can but admire this man for his diligence and the accomplishment in the making of the Tabulae Anatomicae.

Illustrations of accurate anatomical studies are of but recent date, certainly not over 400 years. If comparison is made of the Eustachian, Vesalian, Fallopian and other contemporary anatomical works, with those of the previous decades, one may easily understand the status of surgery and medicine of that period.

Fallopius, Vesalius and Eustachius should be considered an immortal triad, whose life work was of such force that they reformed the study of anatomy into factual and accurate channels by repeated dissections. The acme of their ability was that they were able to illustrate and preserve their dissections by drawings so that the previously accepted inaccuracies were swept away and the true anatomy revealed.

Advances in the Treatment of Cancer of the Larynx

DANIEL S. CUNNING, M.D.
New York, N. Y.

Discussers:

L. B. BERNHEIMER, M.D.
Chicago, Ill.

LeROY A. SCHALL, M.D.
Boston, Mass.

CHEVALIER L. JACKSON, M.D.
Philadelphia, Pa.

There have been many advances made in the treatment of cancer of the larynx since the day in 1873 when Bilroth first removed the larynx for this condition. The courageous surgeons of the nineteenth century had to fight against infection, explosive and fatal hemorrhage, suffocation and pneumonia, esophageal fistula and the profound and hopeless mental depression that haunted the minds of the victims of this dreaded disease.

From 1908 to 1931 St. Clair Thompson and John E. Mackenty perfected the operations of laryngofissure and laryngectomy and did more to alleviate the sufferers of this condition than any other men.

Today we look for almost complete success with the proper treatment of properly chosen cases of cancer of the larynx.

Ninety-nine per cent of all malignancies of the larynx are epidermoid in type. This type of epithelioma is further divided into grades 1, 2, 3 and 4, according to Broders. Experience has shown that a reliable prognosis of any malignancy cannot be based on grading alone but must take into consideration the extent of the growth, its location, vascularity, metastasis, etc. During the past 10 years at the Manhattan Eye, Ear and Throat Hospital 86 per cent of all laryngeal cancer was type 2.

Treatment:

Today there are three accepted methods of treatment of cancer of the larynx.

1. Laryngofissure
2. Laryngectomy
3. Irradiation

Laryngofissure is the ideal treatment of an early epithelioma of one cord. Where the growth crosses over the anterior commissure and involves the opposite cord, I feel that laryngectomy should be the operation of choice. Incipient cancer of the larynx is seldom seen in a hospital clinic.

Laryngectomy. In the clinics of the Manhattan Eye, Ear and Throat Hospital 85 per cent of operations on the larynx are laryngectomies, as we rarely see these cases until they are well advanced and are either extrinsic or show such extensive involvement of the interior that total laryngectomy must be advised.

Irradiation. Improvement in the technic of x-ray therapy in the past few years has done away with many of the bad results of former years, such as sloughing and necrosis of the laryngeal cartilages. But as Doctor Maurice Lenz says, "The treatment still remains a serious undertaking which requires not only familiarity with technical details but also daily clinical control of the laryngeal reaction during treatment." I still feel that in cases suitable for laryngofissure or laryngectomy surgery should be the method of choice, followed by x-ray therapy if necessary.

We as physicians must remember at all times that we are teachers, and the burden of instructing the community on the subject of cancer remains with us. Many agencies of a social nature are active in this educational work and many more have the facilities and are eager and waiting to be used for this purpose. Service clubs, parent-teacher organizations and study clubs will welcome this hopeful news of cancer that can be cured. By ceaseless reiteration to the public of the significance of the hoarse voice and by our constant insistence to physicians of the importance of a mirror examination of the larynx as part of a physical examination, we will soon see the valued results of our efforts. This educational plan will do much to aid in bringing to the laryngologist the incipient case of cancer of the larynx, patients who will qualify for a laryngofissure operation with more than 80 per cent cures.

Labyrinth Surgery for Meniere's Disease

K. M. DAY, M.D., Pittsburgh, Pa.

Discussers:

JOHN R. LINDSAY, M.D.
Chicago, Illinois

MARVIN F. JONES, M.D.
New York, N. Y.

CLARENCE SMITH, M.D.
New York, N. Y.

The author attempts to describe the symptoms and signs associated with hydrops of the labyrinth. He believes that the term "Meniere's Disease" should be limited to this particular class of cases.

The condition may be unilateral or bilateral. These patients suffer from paroxysmal attacks of vertigo, associated with nausea, vomiting, deafness and tinnitus. Patients complain bitterly of the low toned roaring tinnitus. The deafness is of the nerve type with decreased bone conduction, often associated with diplacusis. The hearing varies from time to time. The caloric tests show diminished response from the affected labyrinth, and these responses vary in intensity from time to time.

Many of these patients respond to proper medical treatment. However, there are some who do not and who, in desperation, insist on surgical intervention. It is the author's belief that the nerve section operation is illogical in that it does nothing towards attacking the source of the trouble, namely, hydrops of the labyrinth. Although it does relieve the dizziness, it usually has no effect on the low pitched pounding tinnitus. It is his belief that any surgery in such a case should be upon the labyrinth itself. It should include opening the membranous labyrinth to release some endolymph. He describes an operation for this purpose. By way of the mastoid approach, he opens the horizontal semicircular canal and passes a fine coagulating needle into the vestibule. A report is given of the results of nine cases operated upon by this method, eight of them for hydrops of the labyrinth, and one following a probable hemorrhage into the labyrinth. The lapse of time since operation on six of these cases is from one to three years, so that enough time has elapsed since operation to make some evaluation of the results.

Intrinsic Allergy as It Affects the Ears, Nose and Throat

HENRY L. WILLIAMS, M.D.
Rochester, Minnesota

Discussers:
FRENCH K. HANSEL, M.D.
St. Louis, Mo.
NOAH FOX, M.D.
Chicago, Ill.

A group of symptoms consisting of pains originating in muscles and in blood vessels around the head, and vertigo dependent upon peripheral labyrinthine irritation is a clinical entity on the basis of "physical allergy." "Physical allergy" is a physiologic entity distinct from "antigen antibody." Results of treatment with nicotinic acid are considered and a hypothesis as to the action of nicotinic acid is presented.

Hearing Aids

WALTER HUGHSON, M.D.
Abington, Pa.

Discussers:
JOHN J. SHEA, M.D.
Memphis, Tenn.
GORDON F. HARKNESS, M.D.
Davenport, Iowa

The American Academy of Ophthalmology and Otolaryngology has as one of its appointed committees one which has to do with the Prevention and Amelioration of Deafness and as a subcommittee one concerned with the rehabilitation of the deaf. According to Webster an individual can be rehabilitated by "being restored to a former capacity," in this case hearing, or as a second definition, "to be fit to make one's livelihood again." This discussion will be concerned primarily with rehabilitation.

Medical therapy of deafness is relatively unimportant and ineffective. Surgical treatment if applied according to accepted precepts is from the numerical standpoint relatively insignificant as related to the total problem of deafness and of thirteen million hard of hearing civilian people — children and adults — in the United States.

There were forty thousand aural casualties as a result of World War I out of an army of two million. No one can estimate the aural casualties resulting from this war involving some eleven million men exposed to all the acoustic hazards of modern warfare. Needless to say it will be tremendous.

A completely new field in our specialty is presented both from the civilian and military standpoint. First, from the civilian standpoint every available individual with a hearing handicap must be rehabilitated to carry on some type of war work. Second, it is our responsibility to salvage the aural casualties of the war after they have been discharged from the Armed Forces and the Veteran's Bureau.

How can this be done? On a percentage basis treatment for civilians or war casualties will be numerically insignificant. There is but one sure recourse and that is the use of a hearing aid.

As otologists we have neglected completely this particular phase of our chosen specialty. The time element, equipment and expense are all factors in our general indifference.

Any individual with a hearing loss from an average of 40 decibels to 70 decibels in the speech frequency range can be rehabilitated with a properly fitted hearing aid.

This particular presentation is a plea to otolaryngologists to acquaint themselves with this essential technic of their specialty. It must be recognized that the cases under discussion can be helped and rehabilitated, whether they be civilian or war casualty, only by a hearing aid.

There are patterns of deafness — uniform loss of all frequencies in the audiometric range — predominant loss of the low frequencies, or an emphasis upon the higher frequencies. War casualties will fall predominantly in this latter group.

Ideally, a laboratory with adequate sound equipment should be available for testing the patient's hearing level with and without a hearing aid. Few such centers are available. However, it is essential that every otolaryngologist should be able to test in a relatively simple and completely inexpensive manner the gain obtained by any given hearing aid. An overall gain for speech must be at least 30 decibels regardless of the hearing loss. If this gain is not achieved the aid is worthless.

Methods for testing the gain derived from specific hearing aids will be discussed.

Section on Ophthalmology

WEDNESDAY, OCT. 13, 1943

2 p.m.

RED LACQUER ROOM

Presiding Officer:

SANFORD R. GIFFORD, M.D.

First Vice President

Secretary:

ALGERNON B. REESE, M.D.

The Present Status of Eye Exercises For Improving Visual Functions

WALTER B. LANCASTER, M.D.
Boston, Mass.

Discusser: LAWRENCE T. POST, M.D.
St. Louis, Missouri

Two results are obtained by exercise: (a) Structural changes may result, e.g., hyperplasia and hypertrophy of the muscles; perhaps other structural changes. (b) Function may be improved; greater skill is acquired in the performance of the act which is being practiced.

The pianist after months and years of daily practice has muscles but little larger or stronger but has acquired an enormous amount of skill and coordination, a vast system of conditioned reflexes. Perhaps the structure of the hands has been modified.

In ophthalmology the increase in strength of muscles by exercises has been stressed. This is a grave mistake in emphasis.

Ophthalmologists have not unanimously recognized the value of exercises in improving visual function obtainable by expert application of exercises. This is because they have seen too much of the unskilled and too little of the skilled application of the art of orthoptics.

But the ignorance of many in the matter of orthoptics is as nothing compared to their ignorance of what can be achieved in treating other ocular conditions. This field has been ignored or even avoided by ophthalmologists. The result has been that ignorant people, some of them charlatans, all of them lacking in broad scientific, and especially medical, basic training, have taken it up and exploited it in a way contrary to the best interests of the public.

Data will be given and studied bearing on these problems in the hope of clarifying our ideas and of exciting profitable discussion. As many as possible of the various types and modifications of exercises in use will be taken up, especially those exploited in the lay press. What can be accomplished and how is the good to be accounted for? Have we neglected a means of helping our patients? What harm can be done and how can it be avoided?

Chemical Burns of the Eyes

WILLIAM F. HUGHES, JR., M.D.
Baltimore, Md.

BY INVITATION

Ocular injuries by many types of chemicals produce lesions with similar clinical and pathologic characteristics; e.g., rapid penetration into the ocular tissues with resulting cellular necrosis, a polymorphonuclear response within a few

hours, conjunctival and corneal edema, iritis, vascularization of the cornea within 5-8 days and varying amounts of corneal infiltration and ulceration which are repaired by the proliferation of opaque fibrous tissue.

Acid burns are non-progressive, and the precipitation of the corneal proteins by acid is closely dependent on the character of the anion.

Alkali burns are progressive in type, resembling injuries by vesicant war gases. This relapsing tendency has been attributed to the loss of corneal mucoid, precipitation of calcium salts, the necrosis of limbal blood vessels and the formation of toxic hydrolysates. Treatment consists of (1) immediate removal of residual alkali by irrigations with water or buffer solution, (2) atropine, (3) measures to promote healing of the corneal ulceration and (4) prevention of symblepharon. The beneficial effects of ammonium salts or the emergency removal of necrotic conjunctiva with replacement by oral mucous membrane ("Denig Graft") have not been adequately proven.

Mustard Gas ($ClCH_2CH_2)_2$. A symptomless latent period of 6-36 hours follows exposure to mustard vapor. Liquid mustard is immediately irritating and produces more severe lesions. Although mild ocular burns were common in World War I, devastating or recurrent corneal lesions were rare (0.1% of casualties). Since mustard disappears from the cornea before the victim is aware of having been exposed to mustard vapor, decontamination is impossible. After splash burns, irrigations are of value if performed within 3-5 minutes. Later treatment is directed towards the prevention of secondary complications; e.g., secondary infection.

Lewisite ($ClCH=CHAsCl_2$) liquid or vapor produces excruciating ocular pain and blepharospasm immediately. Compared with mustard, there is greater tissue necrosis and more intense exudation. Treatment consists in the instillation of 2-4 drops of "M 1 Eye Solution" or a small quantity of "M 1 Eye Ointment" immediately after exposure.

N-Mustard resembles Lewisite more than mustard gas, and possesses systemic toxicity. Treatment is symptomatic.

Chloracetophenone vapor produces no permanent ocular damage, the irritation and reflex lacrimation being relieved by local anesthetics.

(The above clinical and pathologic material will be illustrated by lantern slides.)

Some Useful Plastic Procedures in Ophthalmology

GEORGE W. PIERCE, M.D.
San Francisco, California

BY INVITATION

Discusser:
HAROLD F. WHALMAN, M.D.
Los Angeles, California

In this paper will be discussed some particular problems of reconstruction surgery in this field. The problems will be analyzed, and the author's methods of meeting the problems presented. Each type of problem will be accompanied by case history and photographs.

Among the problems to be discussed are the following:

1. Building up of the depressed orbital floor with cartilage, for diplopia.
2. Ectropion.
3. Entropion — A suggested operation.
4. Eyesocket reconstruction with some unusual and difficult reconstructions.
5. Deformities of the lids after enucleation of the globe.
6. Reconstruction of lids after loss by trauma or neoplasm.

The author appreciates his solutions of these problems are his individual reaction and trusts that they will be of value in producing constructive criticism.

A Clear Corneal Implant Acquires Dystrophy from Its Host

ROBERT VON der HEYDT, M.D.
Chicago, Illinois

Discusser: RAMON CASTROVIEJO, M.D.
New York, N. Y.

Eyes which have lost useful vision because of a corneal dystrophy are among the best suited for successful corneal replacements. These eyes are usually normal in other respects. In dystrophy the process involves only part of the clear stromal tissue. This factor enhances the viability of the implant.

My patient, V. K., is now 52 years old. He came to me over 20 years ago suffering from a corneal dystrophy which is now classified as granular disciform, the commonest one of Bueckler's Triad.

I have had his slowly progressing dystrophy under constant slit-lamp observation, as well as photographic recording, during all this time.

His vision 20 years ago was 20/80 —. There occurred a slow gradual increase in the involvement of the central disc area of circumscribed granular bread-crumb-like lesions imbedded within clear stroma. In 1933, 10 years later, because of the progression his vision had been reduced to 10/200 —. Over six years ago I sent him to Doctor Ramon Castroviejo, who successfully transplanted a clear quadratic area of newborn cornea. His vision with correction now is 20/50+.

I noticed a fine sub-surface salt granule-like change in the center of the implant four years ago — two years after the transplantation. After seeing this from time to time the thought occurred to me, what if this implant would acquire the dystrophy from the host? I consoled myself with the thought (this being a definitely hereditary characteristic of the host's cornea), it cannot happen.

This slight change observed for years because of very slow progression has now assumed the definite characteristic picture of the host's dystrophy. There has been no deterioration of the visual acuity because of the invasion of the implant. (Illustrated.)

The Treatment of Recent Injuries of the Eye and Adnexa

MEYER WIENER, M.D.
Coronado, California

Discusser: WENDELL L. HUGHES, M.D.
Hempstead, N. Y.

One must have all facilities necessary in order to do good work in treating injuries in these regions. It may be necessary to evacuate the soldier to the rear of the lines where these facilities may be available. The first principle is to stop bleeding. Tie bleeders with silk ligatures, cut close and leave buried. This should be done after having cleansed the wound thoroughly with soap and water, taking care not to bruise or damage the tissue. It may be necessary merely to check bleeding, counteract shock, cover the eye with a wet dressing and send the patient further back in the lines where the injury can be properly handled.

Give blood transfusion, if necessary. Inflict no unnecessary pain, as this intensifies shock. If needed, give general anesthetic. Rinse the wound after using soap, cut out crushed or ragged tissue, but remove no skin, regardless of how small a piece, as it may be of great value. Give sulfa drugs.

Poor approximation may be worse than none. If the wound is deep, bring deep sutures together first before tying the skin. The outstanding point is to bear in mind that accurate approximation of the wound at the time of first aid may save months of hospitalization, repeated plastic repair and untold pain and suffering at a later date.

A perforating injury always bears suspicion of carrying a foreign body. X-ray examination will determine this. Few intra-ocular foreign bodies are magnetic. Not one has been seen in any of the coast Naval hospitals except those not contracted in actual combat. If one eye has been destroyed and the other has multiple foreign bodies, it may be best not to try to extract any bodies unless they are near the surface and can be easily reached. Magnetic foreign bodies are best removed with a powerful magnet through the sclera. Scleral tears can usually be approximated with conjunctival sutures. If the tear is through the uvea, it is dangerous and must be carefully watched for cells in the chamber. Conjunctival tears usually need no suturing, unless they are extensive. Rupture of the globe usually results in loss of the eye. Scleral ruptures are frequent, often resulting from explosions, and surface manifestations are usually absent. Aneurysm of the cavernous sinus may occur in this manner. Multiple foreign bodies in the cornea and other injuries will be discussed in detail.

The Role of Sarcoid and Brucellosis in Uveitis

ALAN C. WOODS, M.D., Baltimore, Md.
and
JACK S. GUYTON, M.D., Baltimore, Md.

A clinical study of the etiology of uveitis in 562 patients admitted to the wards of the Wilmer Institute between 1926 and July 1, 1939, has previously been reported. The role of sarcoid and of brucellosis as etiologic agents could not be evaluated in that series because the importance of these diseases was not recognized during the period the material was accumulated, and routine tests for sarcoid and brucellosis were not carried out on those patients. This present report is based on a study of patients with uveitis admitted to the Wilmer Institute from July 1, 1939, to July 1, 1943. In this

group of patients sarcoid was looked for by means of routine x-rays of the chest, often x-rays of the long bones, and either biopsy of a lymph gland or determination of the albumin and globulin contents of the blood. Agglutination and complement fixation tests for brucellosis were carried out on these patients, and Brucella skin reactions were investigated when indicated. The importance of sarcoid and of brucellosis as etiologic agents, and criteria for diagnosis, are discussed.

Section on Otolaryngology

WEDNESDAY, OCT. 13, 1943

2 p.m.

GRAND BALL ROOM

Presiding Officer:

PRESIDENT, JAMES A. BABBITT, M.D.

Secretary:

O. E. VAN ALYEA, M.D.

The Development of the Organ of Corti in Relation to the Inception of Hearing

O. LARSELL, Ph.D., Sc.D.
BY INVITATION
EDWARD McCRADY, JR., Ph.D.
BY INVITATION
and JOHN F. LARSELL, M.D.
BY INVITATION
University of Oregon Medical School and the University of the South

Discussers:

ARNOLD A. ZIMMERMANN, Sc.D.
Chicago, Ill.
BY INVITATION

BARRY J. ANSON, Ph.D.
Chicago, Ill.
BY INVITATION

Histological studies of cochleae from developing pouch young of the opossum, tested at various stages for range and sensitivity of auditory response, lead to the following conclusions:

1. Differentiation of the organ of Corti to the functional stage occurs first in the distal portion of the basal coil and the neighboring part of the middle coil in the opossum. From this region it proceeds both in the apical and the basal directions with continued development of the cochlea.
2. As differentiation progresses the cochlea responds to higher and lower tonal ranges. The zones of response to high and low tones appear to correspond roughly to those established by other methods for adult animals and man.
3. There is either an extension or a migration of zones of response to low tones and probably to high tones, as differentiation progresses.
4. The process of differentiation of the pillars and tunnel of Corti is described at various levels of the chochlea in the developmental stages studied, and it is concluded that hearing begins when these structures have attained the necessary degree of development, and that as they differentiate toward the adult stage, the intensity of tone required to produce response decreases. The organ of Corti is interpreted as a tension and cushioning apparatus between the tectorial and basilar membranes which increases the sensitivity of the vibratory mechanism to vibrations of the cochlear fluids.

The Role of Ciliary Action in Production of Pulmonary Atelectasis and Vacuum in Sinuses

A. C. HILDING, M.D.
Duluth, Minnesota

Discusser:

J. KENT LEASURE, M.D.
Indianapolis, Indiana

This report deals with the ciliary mechanism in the diseases of the respiratory tract, and is a continuation of the same study which was reported last year. Last year's paper demonstrated certain pathologic changes in the ciliary mechanism. The present study concerns the role of the intact and functioning ciliary mechanism in the production of pulmonary atelectasis, vacuum headache, and reduced hearing and drum retraction in otitis media.

Pulmonary atelectasis, from the accumulation of quantities of mucous in the air passages, is due to the following mechanical factors:

1. The activity of the ciliary mechanism which forces "mucous plugs" against atmospheric pressure.
2. The tendency of mucous to span air spaces and include quantities of air which are carried out with the mucous.

3. Alternating widening and narrowing of the air passages with respiration.
4. Alternating lengthening and shortening of the air passages with respiration.
5. The bellow-like action of the lungs.
6. The action of cough.

The Laryngeal Node

J. E. ASH, Col., M.C., U.S.A.
Washington, D. C.
BY INVITATION
and LEO SCHWARTZ, M.D.
New York, N. Y.

Discussers:

FRANK J. NOVAK, JR., M.D.
Chicago, Ill.

BERNARD J. McMAHON, M.D.
St. Louis, Mo.

An analysis is made of approximately 125 lesions from the vocal cords, which have accumulated in the Registry of Otolaryngic Pathology under the clinical diagnoses of singers' node, laryngeal polyp, focal pachydermia, varix and "amyloid" tumor. An attempt is made to trace the evolution of the laryngeal node from its origin as an irritation fibroma through the polypoid, varicose and "amyloid" stages. Clinical and pathologic findings are discussed and correlated insofar as is possible with the data at hand.

Anesthesia in Otolaryngology

STUART C. CULLEN, M.D.
Iowa City, Iowa
BY INVITATION

Discussers:

VOSS HARRELL, M.D.
Detroit, Michigan

T. C. GALLOWAY, M.D.
Evanston, Illinois

The primary interest of the surgeon in anesthesia is that it be of such nature that he may complete his work with dispatch and convenience. The primary interest in anesthesia of the patient is that he be spared the discomforts of anesthesia and surgery. The primary interest of the anesthetist is in fulfilling the above demands without increasing the hazard to the patient. The site of surgical manipulations of the otolaryngologist frequently overlaps the area in which the anesthesiologist must also work. Since both are interested in the patient's welfare, it is of paramount importance that the domain of each be in an optimal state.

It is possible, without sacrificing the comfort or safety of the patient, to achieve mutually satisfactory working conditions by the proper application of present day anesthetic agents and methods. Many different agents and technics are now available to the anesthesiologist. With these, which include all inhalation, spinal, intravenous, block, rectal agents and technics, it is usually possible to select a satisfactory solution to the anesthetic problems presented by the otolaryngologist.

The anesthesiologist's service to the otolaryngologist is not confined to the administration of the anesthetic technic mentioned. He may also provide valuable assistance in the sedation of patients, maintenance of airway, oxygen therapy, and in the control of drug reactions, particularly cocaine or similar drugs.

A brief review of the fundamental problems encountered in anesthesia for otolaryngological surgery and a consideration of the possible solution to these problems is presented.

Esophageal Manifestations of Pellagra

GILBERT E. FISHER, M.D.
Birmingham, Alabama

Discussers:

FLETCHER D. WOODWARD, M.D.
Charlottesville, Va.

F. E. Le JEUNE, M.D.
New Orleans, La.

This paper covers a report of 17 patients who presented oral manifestations of pellagra plus the symptom of severe dysphagia. Their dysphagia was so severe that all had been unable to swallow soft or solid food for variable lengths of time. All had lost considerable weight and were anemic. Esophagoscopy in each case revealed a severely hyperemic, intensely dry esophageal mucosa which had from one to seven small ulcerations upon its surface. These patients were given large doses of thiamin chloride, nicotinamide and riboflavin. Dysphagia in each case disappeared within a three month period.

MOTION PICTURES

PLEASE NOTE

(A) New instruments will be shown at the Motion Picture Sessions. Appointments for showing new instruments must be made with the Secretary. In order that proper publicity may be given, members showing a new instrument should prepare a brief description of that instrument and hand this to the Secretary in charge, who will see that it is turned over to the Editorial Board for publication in the TRANSACTIONS.

(B) Exhibitors showing films must acquaint the operator with instructions prior to the time of exhibiting.

(C) Due to legal restrictions on account of fire hazard, only safety film can be shown at the Palmer House at the October convention of the Academy. This applies to 16 mm. and to 35 mm. film, and particularly to sound film. Please be sure that the film you use is safety film.

Motion Pictures, Otolaryngology

MONDAY, OCTOBER 11, 1943

2 p.m.

GRAND BALL ROOM

Presiding Officer:
WESLEY C. BOWERS, M.D.
Second Vice President

Secretary:
O. E. VAN ALYEA, M.D.

FILM NO. 1

Cleft Lip and Palate

T. E. CARMODY, M.D., Denver, Colorado

This motion picture demonstrates the method of closure and technic of relieving tension on flaps with suturing of both mucous membrane flaps and skin of lip.

Palate suturing is demonstrated by a method of split flaps and suturing of nasal and oral surfaces.

FILM NO. 2

Technic of "Narrow Field" Laryngectomy

CHEVALIER L. JACKSON, M.D.
Philadelphia, Pa.
and
CHARLES M. NORRIS, M.D.
Philadelphia, Pa.

BY INVITATION

The recent trend in technic of laryngectomy has been toward a "narrow-field" type of operation, as compared with the "wide-field" type of operation done by the earlier operators. Various types of "narrow-field" technics have been described, but the essential features are simple midline incision, conservation of muscles, overlapping of muscles in closure and simple midline drainage. The skeletonization of the larynx may or may not be done subperichondrially, as advocated by Crowe and Broyles, whose technic must be considered an extreme form of narrow-field operation.

This film shows the narrow-field type of operation which the authors are using at the present time, and in addition to the above mentioned features, it shows the use of a separate circular "buttonhole" incision in the region of the suprasternal notch for the tracheal stoma. A single glass or alloy steel "sump" tube is used for drainage.

FILM NO. 3

Studies of the Physiology of the Cricothyroid Muscle Demonstrated in Motion Pictures

SAMUEL IGLAUER, M.D.
Cincinnati, Ohio
and
KURT TSCHIASSNY, M.D.
Cincinnati, Ohio

BY INVITATION

The action of the cricothyroid muscle has been studied for many years but there is still considerable disagreement as to its exact functions. We have studied the physiology of this muscle on the dog and have taken motion pictures of the responses to electrical stimulation.

As a result of our studies it may definitely be stated that stimulation of the cricothyroid muscle causes the cricoid

cartilage to move upward toward the thyroid cartilage (and not the reverse as stated in some text books). The tensor also is demonstrated.

The respiratory movements of the cricoid cartilage are demonstrated.

The motion picture of these reactions is valuable as a permanent record of the experiments as well as for teaching purposes.

FILM NO. 4

Bomb Blasts and Burns

Bureau of Medicine and Surgery
Navy Department — Washington, D. C.

By means of animation, the types of injuries caused by bomb blasts are described and their first aid measures demonstrated. In bomb blast pictures, the types of burns and their first aid management are described.

FILM NO. 5

A Problem in Communication

JAMES MILTON ROBB, M.D.
Detroit, Michigan

A difficult problem in communication is shown in the development of an artificial voice in a congenital spastic following laryngectomy. With incoordinated movements of head and hands, and spastic contractions of the thoracic cage, it was necessary to develop a dental sound channel and attach the artificial larynx to a headgear. The problem of the spastic thorax was solved by the use of a hand bellows to supply air as an artificial lung.

The dental sound channel is shown in use on a second patient using the artificial larynx in the ordinary manner. The advantage of the device is demonstrated in the improvement of articulation of the labial sounds in speech. Application is suggested in any patient using an artificial larynx.

FILM NO. 6

Kodachrome Cinematography of the Ear, Nose and Throat

PAUL H. HOLINGER, M.D., Chicago, Ill.
and
RALPH G. RIGBY, M.D., Chicago, Ill.

BY INVITATION

Motion pictures of the ear, nose and throat including examination of the ear drum, anterior nasal chamber, posterior nasal space, pharynx, larynx, bronchi and esophagus will be shown. The commoner pathologic lesions of these structures will be illustrated.

FILM NO. 7

Otogenic Chronic Middle Ear Purulencies

SAMUEL J. KOPETZKY, M.D.
New York, N. Y.
RALPH ALMOUR, M.D.
New York, N. Y.
and
JULIUS W. BELL, M.D.
New York, N. Y.

The picture is a dry clinic showing middle ear examinations, the reasons underlying diagnosis and treatment of the chronic suppurations of the middle ear. Both the non-dangerous and the dangerous middle ear chronicities are followed from the patient's first appearance at the clinic to the logical termination of the condition.

The non-operative and the operative groups are separated and demonstrated, and differential factors involved in grouping these facts are augmented by diagrams, animations and laboratory findings. Both operative technic and non-surgical therapy will be discussed and demonstrated.

The film is in technicolor with sound, and it tells its own story.

Motion Pictures, Ophthalmology

TUESDAY, OCTOBER 12, 1943

2 p.m.

GRAND BALL ROOM

Presiding Officer:
WILLIAM A. KRIEGER, M.D.
Third Vice President

Secretary:
ALGERNON B. REESE, M.D.

FILM NO. 1

Ocular Torticollis

FREDERICK C. CORDES, M.D.
San Francisco, California

The film demonstrates the typical muscle findings in a case of ocular torticollis due to a congenital paresis of the left superior oblique muscle. A myomectomy of the left inferior oblique was fol-

lowed by a 4 strand O'Connor cinch on the right superior rectus and a fenestrating tenotomy on the right inferior rectus.

The last half of the film shows the result of the surgical correction.

FILM NO. 2
Iridencleisis and Cyclodialysis
JACK S. GUYTON, M.D.
Baltimore, Md.

Films of these two glaucoma operations are shown to illustrate details believed of importance to the end results.

Iridencleisis: Homatropine two per cent is instilled one-half hour before operation to relax the iris sphincter. Dissection of the conjunctival flap is begun far up on the globe, over the insertion of the superior rectus, to give a large filtering bleb. The episcleral tissue or prolongation of Tenon's capsule is dissected free along with conjunctiva to give a thick covering for the bleb. Dissection of the flap is ended just back of the limbus, avoiding trauma likely to result in undue scarring. A medium sized keratome incision is made into the anterior chamber. The incision is started from two to three millimeters back of the limbus and enters the chamber in the depths of the angle — thus when the iris is included it is pulled upward rather than forward and does not tend to make the chamber more shallow. The iris prolapses as the keratome tip is withdrawn. If it does not prolapse spontaneously, slight pressure on the posterior lip of the wound will cause it to do so. Thus it is unnecessary to insert forceps into the eye. The iris is grasped near its base and the forceps moved gently from side to side in order to tear the root of the iris for two or three millimeters. This procedure rarely causes any bleeding and gives the effect of a small basal iridectomy. DeWecker's scissors are slipped beneath the iris through the resulting tear at the root and the pupillary space. After releasing the iris forceps the iris is snipped vertically. The pillars are folded beneath the conjunctival flap, which is then closed with a running suture. The running suture, not tied at either end, is easy to remove after five to seven days.

Cyclodialysis: This operation is performed between any two recti muscles. It is most satisfactory if performed in the quadrant where the anterior chamber is deepest. Other factors equal, the superior temporal quadrant is chosen because of the ease of exposure and to allow any resulting hyphemia to settle away from the opening when the patient is elevated. A small opening is made through the conjunctiva eight millimeters back of the limbus with scissors and the sclera exposed. An incision is made through the sclera with the point of a Graefe knife. The Randolph cyclodialysis instrument is inserted between sclera and ciliary body into the anterior chamber, the tip being pressed against the sclera to avoid injury to the underlying ciliary body. The instrument is then rotated in an arc of 180°. This instrument has a small hole through it and is attached to a syringe filled with saline. Pressure on the syringe is maintained while the instrument is in the eye, maintaining a firm intra-ocular pressure at all times and thus preventing or greatly reducing anterior chamber hemorrhage. The small conjunctival incision is closed with interrupted sutures, the eye dressed with eserine, and if the operation is performed in an upper quadrant the patient is elevated continuously for 12 hours after operation.

FILM NO. 3
Cataract Extraction Using Corneal Sutures
A. RAY IRVINE, M.D.
Los Angeles, California

The film shows a corneal section and two simple corneal sutures used in cataract extraction. With this method the incidence of postoperative hyphemia, in our hands, has been greatly reduced and, also, the amount of astigmatism. Description of the operation and method is on the film.

FILM NO. 4
Catgut Sutures in Cataract Extractions
WENDELL L. HUGHES, M.D.
Hempstead, New York

This film shows the use of fine 00000 (5-0) gut sutures in cataract extraction with some of the experimental work that was done in rabbits also illustrated. These sutures have now been used routinely for three and a half years and their use has eliminated entirely any complications at the time of the removal of non-absorbable silk sutures with which all ophthalmologists are familiar.

The sutures are not presented as the final word in sutures but as a step in the development of the ideal suture. Experi-

ments were carried out over a period of two years with many different types of suture material before this suture was finally chosen.

FILM NO. 5
Embryology of the Human Eye — An Animated Study
GEORGE H. PAFF, Ph.D., Brooklyn, N. Y.
BY INVITATION

FILM NO. 6
The Mosher-Toti Dacryocystorhinostomy
EDMUND B. SPAETH, M.D.
Philadelphia, Pa.

There has been much written relative to reasons for a dacryocystorhinostomy. In the experience of many persons the operation has but few actual indications, though these are definite. Most other cases of chronic, obstructive, suppurative dacryocystitis are better handled by this procedure —a cystorhinostomy, which obviates the otherwise certain epiphora. The condition is ocular, and the operation should be performed by an ophthalmologist. There seem to be no logical reasons for an intranasal dacryocystorhinostomy.

FILM NO. 7
The Reese Ptosis Operation
EDMUND B. SPAETH, M.D.
Philadelphia, Pa.

This surgical procedure is the ideal operation for congenital ptosis of a high degree and especially when accompanied by a certain amount of epicanthus in early adults wherein levator action is so grossly impaired that the utilization of the levator is impossible. Equally so, it is very satisfactory for a similar type ptosis of high degree in children when superior rectus or levator procedures are impossible of performance. The ptosis of the jaw-winking reflex is also well corrected thereby. Ptosis following the reconstruction of a lid needs only orbicularis fibers for utilization, otherwise the Hess procedure is, in this instance, the better.

FILM NO. 8
Wheeler's Halving Operation
RAMON CASTROVIEJO, M.D.
New York, N. Y.

This film shows patient with small basal cell epithelioma on lower lid excised following Wheeler's technic, leaving rectangular defect on the lower lid which is corrected by the Wheeler halving method without leaving any notching on the lid.

FILM NO. 9
Wheeler's Operation for Correction of Plastic Entropion
RAMON CASTROVIEJO, M.D.
New York, N. Y.

After an incision has been made on the skin parallel to the lower lid margined over the tarsal plate, two strips of the orbicularis are dissected and overlapped; sutured in this way, the stretching of the overlapped orbicularis presses upon the tarsal plate correcting the entropion.

FILM NO. 10
Postoperative Results of Wheeler Ptosis Operation
RAMON CASTROVIEJO, M.D.
New York, N. Y.

Following Wheeler's operation for the correction of congenital ptosis, the operation consists of the attachment of the strips of the orbicularis to the superior rectus.

FILM NO. 11
Gonioscopy
H. S. SUGAR, Captain (MC)
Vancouver, Washington

This film shows the technic for application of the gonioscopic contact glass and the instruments used. The second part shows the chamber angle in a case of aniridia. The last portion shows normal chamber angle during the process of accommodation. During the filming, the other eye of the patient was fixed on test type with a plus 4.00 diopter lens before the eye. The lens was then removed and the changes in the angle during accommodation photographed. After a movement of convergence the ciliary zone of the iris is seen to move slightly forward. Repeated "shots" of this process were taken.

INSTRUCTIONAL SECTION

Officers

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology
A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

THIS SECTION PRESENTS FOR 1943:

1. Thirty-four individual and fourteen sequential ophthalmological courses with a faculty of sixty-seven instructors and covering one hundred and thirty-nine hours.
2. Forty individual and eight sequential otolaryngological courses with a faculty of forty-six instructors and covering one hundred and twenty-seven hours.

Note

We have had many requests for additional courses. The only way in which this could be accomplished during the National emergency was to reduce the length of the instructional periods from one and one-half to one hour in length, and thus establish three instead of two periods each morning.

During the past there has been a great deal of confusion due to late-comers, and if the present system of three periods each morning is to be successful it is imperative that everyone be on time. The instructors will need all the available time for the presentation of their courses. Please remember that the first period begins at 8:30 a.m. instead of 9:00 a.m., and it is suggested that each one be in his seat at least ten minutes before the conference begins.

General Information

Conference Periods

MONDAY, TUESDAY AND WEDNESDAY MORNINGS

($1.50 for each period of instruction)
First Period.....8:30 a.m. to 9:30 a.m.
Second Period.10:00 a.m. to 11:00 a.m.
Third Period..11:30 a.m. to 12:30 p.m.

SEVENTH FLOOR — PALMER HOUSE

The morning periods will be designated with the following key:

M-1.....Monday, First Period 8:30 a.m. to 9:30 a.m.
M-2.....Monday, Second Period 10:00 a.m. to 11:00 a.m.
M-3.....Monday, Third Period 11:30 a.m. to 12:30 p.m.
T-1.....Tuesday, First Period 8:30 a.m. to 9:30 a.m.
T-2.....Tuesday, Second Period 10:00 a.m. to 11:00 a.m.
T-3.....Tuesday, Third Period 11:30 a.m. to 12:30 p.m.
W-1.....Wednesday, First Period 8:30 a.m. to 9:30 a.m.
W-2.....Wednesday, Second Period 10:00 a.m. to 11:00 a.m.
W-3.....Wednesday, Third Period 11:30 a.m. to 12:30 p.m.

(Familiarity with this key will be helpful and will avoid confusion)

General Instructions

Members only who have paid their 1943 dues may make reservations in advance of the meeting. After the session opens no preference will be given to members that is not extended to candidates and guests.

Orders unaccompanied by properly executed checks will not be filled.

Refunds will be granted only if request for reimbursements is received prior to October 7th.

Academy candidates *whose names appear in this issue of the* TRANSACTIONS are exempt from paying registration fee; however, even they are not privileged to reserve tickets in advance of October 10th.

Candidates *whose names do not appear in this* TRANSACTIONS and guests will be required to pay a registration fee of five dollars ($5.00).

All orders for tickets will be filled according to postmark on envelope. Previous to October 1, send orders to 100 First Avenue Building, Rochester, Minnesota; after October 1, mail orders direct to Dr. William L. Benedict, c/o Palmer House, Chicago, Illinois.

Requesting Conference Tickets

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful following of instructions will insure the correct filling of your requests. *Since courses are limited in number it is necessary that you select more courses than it is possible for you to attend; in other words, a list of three preferences for each period for each day should be given.* Please note carefully that where continuous courses are selected the additional periods open in your schedule are varied.

If you are requesting registration in the continuous courses, please specify preferences for individual conferences as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Claiming Conference Tickets

There will be an *Advance Order Registration Section* where members who have sent advance orders will register and secure conference tickets.

Please Note: This year only members in good standing will be accommodated in this way. Members delinquent in dues must make selection of courses in Chicago.

Synopsis of Courses

A synopsis of each course for which a ticket was purchased will be supplied. Please check your assignments at time of registration to see that the proper synopses are in your envelope. As usual, complete sets of synopses or abstracts covering all courses presented may be purchased at the General Registration Desk.

Conference Attendance

There will be a special attendant at the door of each conference room to collect tickets. It is imperative that each registrant have a proper ticket of admittance.

Following is a list of courses and periods. Abstracts of each subject appear after the period assignment.

INSTRUCTIONAL PROGRAM OF COURSES

Continuous Courses

OPHTHALMOLOGY

1943

COURSE 1

Room 752

Periods M-3; T-3; W-3

ARTHUR J. BEDELL, M.D.

Albany, N. Y.

Medical Ophthalmoscopy

A three period course — $4.50

Limited to 45

This will be a continuation of former courses on fundus changes found in constitutional diseases; their significance in diagnosis, prognosis and treatment.

New cases will be illustrated with kodachrome photographs. The common

alterations as well as the rare ones will be presented.

COURSE 2

Room 727

Periods M-3; T-3; W-3
JOHN H. DUNNINGTON, M.D.
New York, N. Y.

Surgery of the Eyelids and Orbit
A three period course — $4.50

Limited to 35

The surgical correction of lacerations, particularly those involving the lid margin and the region of the inner canthus, will be considered, as well as ectropion, coloboma and other anomalies of the lids. Particular emphasis will be placed upon the operative technic and postoperative treatment of various plastic procedures used in the reconstruction of the eyelids.

The surgery of the orbit will include a discussion of (1) Methods of approach for orbital neoplasms, (2) Technic of orbital implantation for the correction of sunken orbit, (3) Methods of restoration of contracted socket.

COURSE 3

Room 796

Periods M 1-2-3; T 1-2-3; W 1-2-3
HARRY S. GRADLE, M.D., et al.

Glaucoma

A nine period course — $13.50

Limited to 50

1. Harry S. Gradle, M.D., Chicago, Ill.
   Classification and Incidence of Glaucoma
2. Peter C. Kronfeld, M.D., Chicago, Ill.
   Physiology of the Balance of Intra-ocular Pressure
3. Georgiana Dvorak-Theobald, M.D., Oak Park, Ill.
   Pathology of Glaucoma
4. Peter C. Kronfeld, M.D., Chicago, Ill.
   Provocative Tests
5. Robert Von der Heydt, M. D., Chicago, Ill.
   Slit Lamp Picture of Chronic Glaucoma
6. Samuel J. Meyer, M.D., Chicago, Ill.
   Chronic Glaucoma — Clinical Picture
7. Thomas D. Allen, M.D., Chicago, Ill.
   The Medical Treatment of Chronic Glaucoma
8. Harry S. Gradle, M.D., Chicago, Ill.
   The Indications for Surgical Interference
9. L. G. Hoffman, M.D., Chicago, Ill.
   Surgery of Chronic Glaucoma

1—Classification and Incidence of Glaucoma

Harry S. Gradle, M.D., Chicago, Ill.

With additional information gained from gonioscopic studies, reclassification of glaucoma is necessary. The terms "primary" and "secondary" are completely discarded and are replaced by "acute" and "chronic."

There will be discussion of incidence of chronic glaucoma according to geographical distribution, race, age, heredity and other influencing factors. The difficulties of collecting this data and the lack of accuracy of such figures will be discussed at length.

2—The Physiology of the Balance of Intra-Ocular Pressure

Peter C. Kronfeld, M.D., Chicago, Ill.

The peripheral mechanisms which are most likely to take effective part in the regulation of the intra-ocular pressure under normal conditions are: the peripheral vasomotor control mechanism, the transfer of intra-ocular fluid out of the eye and the formation of intra-ocular fluid. Of these factors the transfer of intra-ocular fluid out of the eye appears to be the most important one. Through the work of Jonas Friedenwald and also through the advent of gonioscopy, the canal of Schlemm has come into the foreground again and probably represents the main channel of outflow. Friedenwald's concept, that this outflow takes place by virtue of a difference in protein content between the normal contents of the canal and the normal contents of the anterior chamber, postulates a steady inflow of blood plasms into the canal through afferent blood vessels. The possibility of such an inflow has been demonstrated by Friedenwald histologically and by Kronfeld, McGarry and Smith gonioscopically.

3—Pathology of Glaucoma

Georgiana Dvorak - Theobald, M.D., Oak Park, Ill.

The aqueous humor leaves the eye mainly by way of the pectinate ligament,

through the canal of Schlemm into the anterior ciliary veins.

When this exit is occluded, the intra-ocular fluid is increased. This may occur as a result of edema due to a result of some condition causing obstruction to the flow of fluid (primary glaucoma), or to some more obvious mechanical obstruction of the exit angle (secondary glaucoma).

Whether the glaucoma is primary or secondary, the changes occurring in the various tissues are similar and are due to edema and pressure.

4—Provocative Tests

Peter C. Kronfeld, M.D., Chicago, Ill.

The discussion will include the more commonly employed, as well as some of the more uncommon, provocative tests. Special emphasis will be placed on the relation of the various tests in relation to the chamber angle depth.

5—The Slit Lamp Picture of Chronic Glaucoma

Robert Von der Heydt, M.D., Chicago, Ill.

The discovery that lamellar exfoliation of the senile sclerosed lens capsule is the cause of glaucoma is one of the most important contributions of microscopy of the living eye to ophthalmic diagnosis.

The exfoliated capsular debris can be easily seen on the iris margins and within the anterior chamber. Its interference with normal ocular drainage causes an increase in intra-ocular tension.

About 9 per cent of the cases of simple glaucoma are due to this condition. A disclosure of its incipience is one of the many valuable contributions of the slit lamp in the field of ocular diagnosis.

6—The Clinical Picture of Chronic Glaucoma

Samuel J. Meyer, M.D., Chicago, Ill.

The various symptoms in compensated glaucoma will be elaborated in the order of their importance. Discussion of the objective findings will include colored lantern slides of the optic nerve changes, tonometric technic, etc. Methods of examining the visual field changes will be particularly emphasized. The importance of an early diagnosis will be stressed.

7—The Medical Treatment of Chronic Glaucoma

Thomas D. Allen, M.D., Chicago, Ill.

The symptom complex called glaucoma is too casually considered by the medical profession. The diagnosis, especially in the compensated types, is very often overlooked and when made, two conclusions are immediately drawn concerning treatment — operation or miotic agents.

Each individual case should be carefully studied in its relationship to the general condition and to the effect of various drugs and physical agents upon it. Eserine and pilocarpine are no longer the only drugs and often are not the wisest drugs to use. The physical means of reducing the tension and of promoting better intra-ocular health are often completely ignored.

An attempt will be made to review this entire subject.

8—The Indications for Surgical Interference

Harry S. Gradle, M.D., Chicago, Ill.

9—The Surgery of Chronic Glaucoma

L. G. Hoffman, M.D., Chicago, Ill.

The preparation of the patient, anesthesia and selection and care in instruments will be discussed. The technic of cyclodialysis, trephine and the inclusion operations are given in detail. The prevention of accidents and complications with their correction is dwelt upon.

The reasons for failure and expectation of results with a summary of statistics from the literature is included.

Motion pictures of the above mentioned operations will be shown.

COURSE 4

Room 734
Periods M 2 & 3; T 2 & 3; W 2 & 3
LeGRAND H. HARDY, M.D., et al.

Orthoptics
A six period course — $9.00

Limited to 35

1. LeGrand H. Hardy, M.D., New York, N. Y.
   Orthoptics
2. Truman L. Boyes, M.D., New York, N. Y.
   Management of Orthoptic Therapy
3. George P. Guibor, M.D., Chicago, Ill.
   The Value of Orthoptics in Private Practice.

1—Orthoptics

LeGrand H. Hardy, M.D., New York, N. Y.

1. Definition of orthoptics.
2. Orthoptic training and surgery.
3. Misapplications.
4. The safe approach to orthoptics.
5. Orthoptics and the heterophorias.
6. Subjective versus objective cures.
7. Orthoptics and tropias.
8. Dangers and safeguards.
9. The American Orthoptic Council.

2—Management of Orthoptic Therapy

Truman L. Boyes, M.D., New York, N. Y.

An abbreviated history of orthoptics and the practical discussion of clinic organization and associated home treatment will be given. The routine management of visual and orthoptic therapy in children and adults will be discussed. There will be an analysis of statistics showing the final results and benefits between patients who had orthoptic therapy and those who had no orthoptic therapy.

3—The Value of Orthoptics in Private Practice

George P. Guibor, M.D., Chicago, Ill.

An orthoptic routine should consist of a diagnostic and therapeutic procedure and of these two, the former is the more important.

In the diagnostic routine, the screen or cover test is considered the most valuable procedure used in determining the presence or absence of a motility defect. This test, therefore, will be discussed in detail.

Following the diagnosis of the type of deviation and the determination of the etiology, the therapeutic routine may include the correction of refractive errors, the use of prisms, cycloplegic agents, occlusion and fusion exercises with or without surgery. The cause of the deviation found in heterophorias or heterotropias should be determined if possible because the etiology in such a case often suggests the treatment to be employed.

Case histories of the types of strabismus seen by the oculist in infants, in youths and in older patients will be discussed from a neurologic standpoint.

COURSE 5

Room 731

Periods M-2; T-2

WENDELL L. HUGHES, M.D.

Hempstead, N. Y.

Surgery of the Lids

A two period course—$3.00

Limited to 30

A few of the common lid conditions will be discussed paying particular attention to traumatic conditions, lacerations, ectropion, entropion and lid reconstruction.

COURSE 6

Room 727

Periods M-1; T-1; W-1

WALTER B. LANCASTER, M.D.

Boston, Mass.

Practical Physiological Optics

A three period course—$4.50

Limited to 35

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(1) An exposition of the real facts of image formation in the eye and their bearing on the daily work of the refractionist, brushing aside nodal points, principal planes, equations, etc., as "so much useless ballast." (Gullstrand).

(2) An exposition of accommodation, its nature, how it can be controlled—the pitfalls of cycloplegic vs. the pitfalls of non-cycloplegic refraction.

COURSE 7

Room 731

Periods M 1 & 2; T 1 & 2; W 1 & 2

DONALD J. LYLE, M.D.

Cincinnati, Ohio

Neuro-Ophthalmology

A six period course — $9.00

Limited to 40

This course will include a discussion of the anatomy and physiology of the central nervous system with pathological application in reference to ophthalmology.

A description will be given of the visual system from the retina through the conductive paths to the visual, sensory and psychic areas of the brain.

The voluntary and autonomic motor system of the eye will be discussed.

Special attention will be given to a discussion of reflexes and syndromes concerned with ophthalmology.

COURSE 8

Room 740

Periods M 1 & 2; T 1 & 2; W 1 & 2

ROBERT J. MASTERS, M.D., et al.

Slit-Lamp Microscopy

A six period course — $9.00

Limited to 25

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1. Robert J. Masters, M.D., Indianapolis, Ind.
2. Harvey E. Thorpe, M.D., Pittsburgh, Pa.
3. Peter C. Kronfeld, M.D., Chicago, Ill.

The lectures and demonstrations of this course are planned for beginners in slit-lamp microscopy and for those registrants who desire a review of the elementary features of biomicroscopy.

The early part of each session is given over to an illustrated lecture, which is followed by a longer period of actual work with the slit lamps by the members of the class.

The illustrated lectures cover, in regular order from day to day, studies of the normal and pathologic cornea, anterior chamber, iris, crystalline lens and vitreous humor. Special attention is given to the findings which can be made with no other instrument, and which make the slit lamp indispensable in the routine practice of ophthalmology.

The demonstration period is utilized to make each class member familiar with the manipulation of the instruments, several of which are available in the class room. During each demonstration period a round table question-and-answer discussion is carried on for the class members who are already familiar with the instruments.

COURSE 9

Room 738

Periods M 1-2-3; T 1-2-3; W 1-2-3

AVERY de H. PRANGEN, et. al.

Refraction

A nine period course — $13.50

Limited to 50

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1. F. W. Jobe, B.S., Bausch & Lomb, Rochester, N. Y.
   BY INVITATION
   Practical Physiological Optics
2. John G. Paul, Guild Optician, Rochester, N. Y.
   BY INVITATION
   Dispensing and Frame Fitting
3. Scott Sterling, A.B., Bausch & Lomb, Rochester, N. Y.
   BY INVITATION
   The Selection and Application of Lenses Used in Ophthalmology
4. Avery de H. Prangen, M.D., Rochester, Minnesota
   Characteristics, Management and Prescribing for Various Refractive Types
5. Ralph H. Woods, M.D., La Salle, Ill.
   Synopsis of Subjective Refraction by the Use of Concave Cylinders. Subjective Testing.
6. Hendrie W. Grant, M.D., St. Paul, Minn.
   Accommodation — Convergence and Divergence
7. Donald O'Rourke, M.D., Denver, Colo.
   Skiascopy, Cross Cylinders and Use of Cycloplegia
8. James J. Regan, M.D., Boston, Mass.
   Manifest and Fogging
9. Summary and discussion.

1—Practical Physiological Optics

FREDERICK W. JOBE, B.S.

Rochester, N. Y.

BY INVITATION

The function of an optical system, whether it is a spectacle lens or a range-finder, consists of optically moving a physical object into a position where the object can be used. In refraction this position is the far point of the eye. The general laws governing the performance of a lens or prism are readily understandable. A knowledge of these principles is extremely valuable in the practice of refraction.

These general laws will be discussed in relation to refraction, effective power, transposition of formulae, spherical equivalents, cross cylinders and Sturm's interval, as well as some ophthalmic instruments such as refractors, duochrome tests and keratometers.

2—Dispensing and Frame Fitting

John G. Paul, Guild Optician, Rochester, N. Y.

BY INVITATION

Dispensing in its different phases will be discussed. Procedures for taking a complete set of measurements for glasses and all information in regard to their intended use will be outlined. Determination of the size, shape and type of lens, also the size of the bridge and length of the temples will be considered. Demonstration: Adjustment of glasses to hold proper position with comfort.

3—The Selection and Application of Lenses Used in Ophthalmology

Scott Sterling, A. B., Rochester, N. Y.

BY INVITATION

In this course a review will be given of the significant optical characteristics of the various types of single vision and bifocal lenses, with a brief discussion of the advantages and disadvantages of each. Emphasis will be placed upon the practical problem of establishing criteria by which the ophthalmologist may select suitable lens types for specific cases.

4—Characteristics, Management and Prescribing for Various Refractive Types

Avery de H. Prangen, M.D., Rochester, Minn.

The themes of contrast and balance as they apply to subjective testing and prescribing in refraction will be discussed. Some of the aspects of the correction of the spherical part of hyperopia, myopia and mixed astigmatism will be cited, together with indications and methods for partial correction of high degrees of astigmatism and the handling of presbyopia. Slides illustrative of unusual refractive errors and their correction will also be shown.

5—Synopsis of Subjective Refraction and the Use of Concave Cylinders. Subjective Testing.

Ralph H. Woods, M.D., La Salle, Ill.

The practical application of the finer points in subjective testing will be discussed. A well done pre-cycloplegic refraction is imperative in order to know the punctum remotum, the punctum proximum, the amplitude of accommodation and its relation to convergence. Technics of subjective testing will be stressed including the use of minus cylinders.

6—Accommodation — Convergence and Divergence

Hendrie W. Grant, M.D., St. Paul, Minn.

Accommodation will be discussed from the standpoint of physiology, mechanism and theory of production, range of accommodation, amplitude, change with age, physical and physiological accommodation, the phenomena associated with accommodation, association of convergence and accommodation, monocular and binocular accommodation and the anomalies of accommodation.

Convergence will be discussed from the standpoint of methods of testing, the norms of convergence, convergence near point and convergence adduction, the anomalies of convergence, convergence excess and convergence insufficiency. The treatment of anomalies and cases illustrating the above will be cited. Methods used in prescribing will be outlined.

7—Skiascopy, Crossed Cylinders and the Use of Cycloplegia

Donald H. O'Rourke, M.D., Denver, Colorado

The various strengths of the crossed cylinders in the subjective testing of refraction are a very important aid. Many refractionists consider them indispensable. Their practical application will be explained and demonstrated. The theory and practice of skiascopy (retinoscopy) with cylinders will be demonstrated. The similarity and relationship of subjective testing with crossed cylinders and objective testing by retinoscopy will be discussed. The use of cycloplegia in refraction will be discussed.

8—Manifest and Fogging

James J. Regan, M.D., Boston, Mass.

The hour will be devoted to the actual demonstration of dependable technics for measuring errors of refraction subjectively, the only exception being the use of the retinoscope. Fogging will be demonstrated, and the theory as well as practical application will be discussed during the demonstration.

9—Summary and informal discussion by the teachers and the participants in this course.

COURSE 10

Room 726
Periods M 2 & 3; T 2 & 3; W 2 & 3
EDMUND B. SPAETH, M.D.
Philadelphia, Pa.

Plastic Repair of Lid Defects and Defects of the Orbit
A six period course—$9.00

Limited to 30

The correction of defects of the lids and the orbit will be discussed by means of slides, drawings and motion picture films. The various defects should be considered on a basis of etiology, degree of tissue loss and the different methods available for the correction under consideration.

COURSE 11

Room 733
Periods M 1 & 2; T 1 & 2; W 1 & 2
GEORGIANA DVORAK-THEOBALD, M.D., et al.

Pathology
A six period course — $9.00

Limited to 35

1. Brittain F. Payne, M.D., New York, N. Y.
   Anatomy and Histology of the Eyeball.
2. John M. McLean, M.D., New York, N. Y.
   Retinal Tumors.
3. Alson E. Braley, M.D., New York, N. Y.
   Glaucoma.
4. Georgiana Dvorak-Theobald, M.D., Oak Park, Ill.
   Retinopathy in Vascular Disease.

COURSE 12

Room 722
Periods M-3; T-3; W-3
JAMES W. WHITE, M.D.
New York, N. Y.

Ocular Motor Anomalies
Extension Course
A three period course — $4.50

Limited to 40

In the three periods assigned to this course, the physiological action of the extra-ocular muscles will be reviewed and the principles of the pathological variations developed. Tests will be considered with their uses, interpretations and limitations.

The non-surgical and surgical treatment will be discussed.

COURSE 13

Room 722
Periods M 1 & 2, T 1 & 2, W 1 & 2
JAMES W. WHITE, M.D., et al.

Anomalies of the Extra-Ocular Muscles
A six period course — $9.00

Limited to 40

1. James W. White, M.D., New York, N. Y.
   Non-Operative Treatment and the Relation of Ametropia to Motor Anomalies.
2. Rudolf Aebli, M.D., New York, N. Y.
   Congenital Anomalies.
3. Rudolf Aebli, M.D., New York, N. Y.
   Physiology and Pathology of the Extra-Ocular Muscles.
4. John H. Dunnington, M.D., New York, N. Y.
   The Surgical Treatment of Heterophoria and Heterotropia.
5. Maynard C. Wheeler, M.D., New York, N. Y.
   Tests
6. Maynard C. Wheeler, M.D., New York, N. Y.
   Tests

1—Non-Operative Treatment and the Relation of Ametropia to Motor Anomalies
James W. White, M.D., New York, N. Y.

In considering the various methods of non-surgical treatment of the phorias and tropias, a definite differential diagnosis must be made. All treatment depends on the proper estimation of the various points in the diagnosis. Treatment will include the use or non-use of glasses for the various types of deviations and the indications for prismatic correction of the various phorias and tropias. The principle of orthoptic training will be discussed as to the results to be expected from their use in the different types of anomalies.

2—Congenital Anomalies

Rudolf Aebli, M.D., New York, N. Y.

The subject matter will cover the more usual congenital anomalies such as retraction syndrome and its variations, strabismus fixus, Marcus Gunn or jaw winking syndrome, paralyses of both elevators of one eye with true and pseudo-ptosis and also congenital individual muscle anomalies.

3—Physiology and Pathology of the Extra-Ocular Muscles

Rudolf Aebli, M.D., New York, N. Y.

The physiological actions of the ocular muscles will be discussed.

a. As individual muscles.
b. In the acts of associated movements.
c. In the disjunctive acts of convergence, divergence, sursumvergence and rotations.

From these physiological acts, the pathological variations from normal are developed and classified, and a method of differential diagnosis outlined.

4—The Surgical Treatment of Heterophoria and Heterotropia

John H. Dunnington, M.D., New York, N. Y.

Special emphasis will be placed upon the importance of selecting the proper type of operative procedure. A brief review of the methods used to aid in this selection will then be given, followed by a full discussion of the indications and contra-indications for the different types of surgical procedures.

5 and 6—Tests

Maynard C. Wheeler, M.D., New York, N. Y.

This will include a demonstration and discussion of the most important methods used in the diagnosis and measurement of muscle defects. Particular emphasis will be placed on the screen test and its actual application in routine practice.

COURSE 14

Room 732 **Periods: M-2; T-2**

ALAN C. WOODS, M.D.
Baltimore, Md.

Ocular Syphilis
A two period course—$3.00

Limited to 40

Syphilis of the eye is considered in relation to the type and stage of syphilis in which the ocular lesions most frequently occur. Under congenital syphilis, optic neuritis, choroiditis and interstitial keratitis are discussed. Under early acquired syphilis, iritis, choroiditis and the early neuro-lesions are reviewed. Under late acquired syphilis, the gummatous and late inflammatory lesions, syphilis of the orbit, the neuro-lesions of late chronic meningovascular syphilis and the degenerative nerve lesions are considered.

Continuous Courses

OTOLARYNGOLOGY

1943

COURSE 51

Room 727 **Periods: M-2; T-2; W-2**

LOUIS H. CLERF, M.D.
Philadelphia, Pa.

Causes of Hoarseness with Particular Reference to Treatment
A three period course — $4.50

Limited to 30

Hoarseness, a symptom, always should direct attention to the larynx. The mechanism of laryngeal voice production and the clinical significance of hoarseness will be discussed. The differential diagnosis of causes of hoarseness and the treatment indicated will be described.

COURSE 52

Room 744 **Periods: M-3; T-3; W-3**

WALTER HUGHSON, M.D., Abington, Pa.

Audiometric Technics and the Fitting of Hearing Aids
A three period course — $4.50

Limited to 40

This course will involve a consideration of the methods of testing hearing and making accurate diagnoses by means of modern electric audiometric equipment. It is felt that all functional tests of hearing can be carried out with much greater accuracy by such methods than by the conventional and extremely inaccurate tuning fork, watch tick and spoken voice method.

The theory and practice of audiometry will be discussed in detail. Special tests

such as "Loudness Balance" and "Speech Reception Thresholds" will be described. The specific application of auditory masking will be explained. Group audiometry, modern methods of exposing malingering and finally the interpretation of audiograms will all be considered.

The fitting of hearing aids is an exact science. The entire field of hearing aid characteristic as related to specific hearing loss will be analyzed. Psychologic and occupational factors will be evaluated as well as what a properly fitted hearing aid must be expected to do for the individual with impaired hearing. The performance of specific aids both in relation to air and bone conduction and make will be dealt with on the basis of patient approval.

COURSE 53

Room 750 Periods: M-1; T-1

MARVIN F. JONES, M.D.
New York, N. Y.

CAUSES OF DIZZINESS

A two period course — $3.00

Limited to 30

Thirty-five causes of dizziness will be discussed. Other causes could be added but the ones named are definite. The conference will be conducted as a basic science course plus a clinical hour. The first day will be spent in an exposition of the mechanisms involved together with the pathology and physiology of the equilibratory apparatus. The second period will be utilized discussing the establishment of a diagnosis in those patients whose symptoms include dizziness. The recognized and theoretical treatments used today will be given in detail. Some unpublished results of this work will also be presented.

COURSE 54

Room 732 Periods: M-3; T-3; W-3

V. H. KAZANJIAN, M.D., Boston, Mass.

Early Treatment of War Injuries of the Face and Jaws

A three period course — $4.50

Limited to 35

First, the general nature of war injuries of the face and jaws, including fractures of the facial bones and lacerations of the soft tissues, will be discussed. Second, a few pertinent points in diagnostic examination of fractures and lacerations of soft tissues will be emphasized. Third, the importance of early treatment will be shown with illustrations. Finally, a discussion of various complications arising in these conditions will be outlined.

COURSE 55

Room 732 Periods: M-1; T-1; W-1

J. D. KELLY, M.D., New York, N. Y.

Surgical Treatment of Bilateral Paralysis of the Abductor Muscles of the Larynx

A three period course — $4.50

Limited to 35

The purpose of this course is to acquaint the students with the different surgical procedures that have been devised for the relief of bilateral abductor paralysis of the larynx following injuries and diseases; also to mention the mechanical results achieved by each type of operation and the application of these operations to the various patients.

A review is also made of the anatomy and physiology of the larynx as it pertains to this type of surgical procedure.

COURSE 56

Room 732 Periods: M-3; T-3; W-3

SAMUEL J. KOPETZKY, M.D.
New York, N. Y.

What Can Be Done for the Deafened

A three period course — $4.50

Limited to 35

The first hour will be devoted to a consideration of the physiology of hearing, the microphonetics of the ear and the basis for the diagnostic tests for hearing. Stress will be laid on the different categories into which the tests separate the patients.

The second lecture will be concerned with the selection of specific cases and the indications for specific therapy and the reasons why the specific therapy might reasonably be expected to improve the hearing. The management of the deafened patient, from early childhood to advanced age, will be discussed, and the necessity for managing the case, in addition to therapeutically handling it, will be brought out.

The third lecture will embrace the subject of surgical therapy, with a review of the various technics which have been advocated to date. The advantages and disadvantages which each type of surgery holds and a discussion of results obtainable from various technics will also be covered. The lecture will conclude with a general summation of the answer to the question of what can be done for the deafened.

COURSE 57

Room 744 **Periods: M-2; T-2**

SCOTT N. REGER, Ph.D.
Iowa City, Iowa

BY INVITATION

Audiometry and Hearing Aids
A two period course — $3.00

Limited to 40

I. Audiometry.
   A. Technic of presentation of stimulus and recording of results.
   B. Bone conduction acuity measurement.
   C. Factors influencing accuracy of measurements; masking and noise.
   D. Interpretation of results.

II. Hearing Aids.
   A. Types of instruments.
   B. The audiogram in relation to the fitting of hearing aids; the minimum and maximum losses of hearing at which hearing aids cease to be effective.
   C. Speech intelligibility and articulation comparison tests.
   D. Psychological factors influencing the patient's reaction to hearing aids.

COURSE 58

Room 750 **Periods: M-3; W-3**

LYLE M. SELLERS, M.D., Dallas, Texas

Equilibrium and Vertigo
A two period course — $3.00

Limited to 30

Disturbances of equilibrium apparently constitute a difficult and complex problem. But the difficulty and the complexity are more apparent than real. This course is designed to clarify and to simplify the problem and to assist the otologist in correctly diagnosing the underlying condition or conditions producing the symptoms, whereby effective therapeutic measures may be applied.

The clinical tests of the vestibular apparatus are presented, explained and amply illustrated by means of both still projections and by movies. But first the anatomy and the later and more correct conceptions of the physiology of the labyrinth (including the cochlea) considered as an anatomico-physiologic unit of the central nervous system are presented in such a manner as to simplify and to clarify the interpretation of these clinical tests. Here will be included an apparently otherwise unpublished observation on the differential diagnosis of high and low lying central lesions.

Case histories illustrating both the simpler and the more difficult problems, together with the solutions of these problems, will be presented.

Individual Courses
OPHTHALMOLOGY
1943

COURSE 201

Room 724 **Periods M-2; T-2**

A. W. ADSON, M.D., Rochester, Minn.

BY INVITATION

The Differential Diagnosis and Treatment of Neuralgias of the Face

The results accomplished in the treatment of facial pains is in direct proportion to the accuracy of the diagnosis and the appropriate treatment instituted. The two major neuralgias are trigeminal and glossopharyngeal involving the fifth and ninth cranial nerves respectively. The attacks of pain are similar though the areas involved differ. Both neuralgias are cured by intracranial section of the nerve affected. Alcohol injected into the peripheral branches of the fifth nerve results in temporary relief. This procedure is not suited for glossopharyngeal neuralgia because of the close proximity of the vagus nerve. The treatment of other facial pain is designed to relieve the particular cause.

COURSE 202

Room 751 **Periods T-1; W-1**

BENNETT Y. ALVIS, St. Louis, Mo.

Acute Corneal Lesions — Diagnosis and Treatment

1. Therapeutic agents: Anesthetics, antiseptics, stimulants, cauterants. Surgery.
2. Injuries: Foreign bodies, abrasions, lacerated and incised wounds, infections, burns (thermal, chemical, radiation), exposure keratitis.
3. Inflammations:
   Ulcers—simple, hypopyon, ring, Mooren's.
   Virus diseases—herpes, simplex and zoster; dendritic ulcers; superficial punctate keratitis.
   Keratitis filamentosa, phlyctenular disease, avitaminosis, interstitial keratitis.
4. Fever therapy.

COURSE 203

Room 705 **Periods M-2; T-2**

W. S. ATKINSON, M.D., Watertown, N. Y.

Anesthesia Used in Ophthalmology

A comparison of the more frequently used local anesthetic agents will be given.

There will be illustrations and descriptions of methods to produce local anesthesia.

The indications for general anesthesia will be considered with a brief discussion of general anesthetic agents and the methods of administration suitable for eye surgery.

COURSE 204

Room 777 **Periods M-2; T-2**

CHARLES A. BAHN, M.D.
New Orleans, La.

The Visual Requirements in the Armed Services

Visual requirements as well as qualifying and disqualifying conditions and diseases in all branches of the military services will be presented. Included will be visual requirements of enlisted and commissioned personnel, male and female, in the Selective Service, Army, Navy, Marine Corps, Coast Guard, U. S. Public Health Service and the Air Services.

Test methods used by the Army and Navy will be described, and information of interest to military ophthalmic examiners will be presented.

COURSE 205

Room 744 **Period M-1**

S. JUDD BEACH, M.D., Portland, Maine

Refinements of Refraction — Problems of Low Myopia

On account of assumed immunity to eye-strain, symptoms due to low myopia are apt to be overlooked. Similarly there are complications of presbyopia peculiar to low myopia. These and other aspects of low nearsightedness, like the supposed inability to obtain 20/20 vision without lenses, will be discussed in this conference.

COURSE 206

Room 706 **Periods M-3; T-3**

CONRAD BERENS, M.D.
New York, N. Y.

The Etiology and Treatment of Uveitis

Certain experimental considerations of the etiolgoy of uveitis will be discussed, especially from the standpoint of animal inoculation. The clinical aspects of the etiology will be presented with statistics of various investigators, including recent statistical studies of private and hospital patients in the New York area. The clinical diagnosis will be considered in relation to pathologic findings. Various methods of treatment will be discussed from the standpoint of the suspected underlying etiology of the uveal inflammation.

COURSE 207

Room 702 **Periods M-3; T-3; W-3**

PAUL BOEDER, Ph.D.
Southbridge, Mass.

BY INVITATION

Aniseikonia

A clarification of the concept of aniseikonia will be followed by a description of the means of measurement and correction. Some statistics will be presented. The effect of aniseikonia on binocular space perception will be discussed in detail, and the possibility of a functional test of aniseikonia will be outlined.

COURSE 208

Room 736 **Periods T-2; W-2**

ALBERT L. BROWN, M.D.
Cincinnati, Ohio

Ocular Tuberculosis

COURSE 209

Room 706 **Periods M-1; T-1**

FRANK E. BURCH, M.D., St. Paul, Minn.

End Results in Retinal Detachment

What types of cases are most favorable for successful operation for retinal detachment?

What is a good result and what must one consider a failure in reporting results?

The comparative results in patients with myopia, high myopia, previous fundus pathology, post-trauma, aphakia and other complications will be reviewed. Also, the results incident to age of patient at time detachment occurs, extent of detachment, various types and location of tears or holes, or disinsertions, will be discussed.

The type of operation done, variations in technic and reasons for redetachment are considerations which affect the "end results."

COURSE 210

Room 744 **Periods T-1; W-1**

FRANK D. CARROLL, M.D.
New York, N. Y.

OPTIC NEURITIS

In this lecture will be discussed all the inflammations of the optic nerve — the differential diagnosis, etiology and recent advances in treatment. A practical method for determining the possible causes of optic nerve pathology will be presented, and consideration will be given to the toxic amblyopias, especially those which may become more frequent and important during the present World War.

COURSE 211

Room 720 **Periods T-3; W-1**

RAMON CASTROVIEJO, M.D.
New York, N. Y.

Corneal Plastic Surgery

In this course the following subjects will be treated: Conjunctival keratoplasty and other plastic operations for recurrent pterygium, pseudo-pterygium and symblepharon; operations for conical cornea; tatooing of the cornea; superficial keratectomy for the removal of corneal scars; superficial corneal dystrophies and pannus; surgical treatment of corneal staphyloma and keratoplasty.

COURSE 212

Room 718 **Periods M-2; T-2**

ALFRED COWAN, M.D.
Philadelphia, Pa.

Presbyopia and Complicated Lens Prescriptions

Presbyopia will be defined. The onset of presbyopia and correction dependent on individual characteristics and requirements will be discussed. Methods of refraction in young patients with presbyopia will be considered as well as modern ophthalmic lenses, bifocals and choice of shape and type of lenses. The importance of adjustment of glasses will be taken into consideration.

COURSE 213

Room 754 **Periods M-1; W-1**

WILLIAM H. CRISP, M.D., Denver, Colo.

Cross Cylinders

1. Principles and technic of cross cylinders for astigmatic axis and strength
2. Sources of error in testing for strength of cylinder.
3. Coordination of astigmatic tests with use of fogging method.
4. Use of strong cross cylinders for amblyopic eyes.
5. Relation between cross cylinders and astigmatic dials as to the cylinder axis and strength.

COURSE 214

Room 778 **Periods M-2; T-2**

LOUISE FARWELL DAVIS, Ph.D.
Director, Child Guidance, National College of Education
Evanston, Illinois
BY INVITATION

Eye Defects and Their Effect in Faulty Reading Habits in the Problems of the School Child

This course will cover, by means of case records, a discussion of children experiencing these conditions:

1. Farsightedness and its connection with
   A. Poor visual imagery
   B. Dislike for reading
   C. Visual discomfort and fatigue
2. Poor motor control of eyes.
3. Inadequate focusing and low fusion range with their resulting problems in reading and spelling.

COURSE 215

Room 713 **Periods M-3; T-3**

HAROLD F. FALLS, M.D.
Medical Director, Department of Human Genetics, University of Michigan
Ann Arbor, Mich.

BY INVITATION

Hereditary Aspects of Ophthalmological Conditions

This is an introduction to the genetics of common hereditary ocular anomalies. Primary principles of heredity will be briefly discussed and demonstrated. Illustrations and slides of the ocular dyscrasias will accompany the didactic subject material.

An outline of information requisite to accurate prognostication of the inheritance pattern of numerous ocular defects will be presented.

COURSE 216

Room 716 **Periods M-3; T-3**

W. GUERNSEY FREY, JR., M.D.
New York, N. Y.

Office Ophthalmology

The art of conducting an ophthalmological practice and office is not learned by taking a residency in special courses. The technic and business side of the profession will be discussed with specific hints for improved relations with patients and fellow practitioners. The entire period will be informal and an exchange of views and questions will be encouraged.

COURSE 217

Room 713 **Periods M-1; T-1**

A. D. FROST, M.D., Columbus, Ohio

External Diseases of the Eye

The best way to deal with so broad a subject is to concentrate on some common errors in diagnosis and treatment of the more usual external diseases as observd in ophthalmic consultations. Clinic discussion from the group will be welcomed.

COURSE 218

Room 721 **Periods M-2; W-2**

GLEN G. GIBSON, M.D.
Philadelphia, Pa.

The Correlation of the Etiology and Surgical Treatment of Paralytic and Concomitant Strabismus

The various central and peripheral causes of paralytic strabismus will be discussed and compared to the causes of concomitant strabismus. A system of graduating the degree of abnormal muscle action will be presented. The method for deciding when, where and how much to do will be based upon the evaluation of the motor and sensory disturbances.

COURSE 219

Room 750 **Periods T-2; W-1**

DAVID F. GILLETTE, M.D.
Syracuse, N. Y.

Degeneration of Vitreous — Etiology and Treatment

The plan of the course is to give a review of the development, the present concept of the structure and the metabolism of the vitreous body. There will be considered the changes that occur in the vitreous in the presence of myopia, in the presence of disease of the neighboring tissues and in the aged without any visible pathology of these tissues. Special attention will be given to the study of vitreous opacities.

COURSE 220

Room 739 **Periods M-1; T-1**

OTTO GLASSER, Ph.D., Cleveland, Ohio

BY INVITATION

Defects of Color Vision—Types and Tests

A brief discussion of the theories of color vision and a short history of observations of defects of color vision will be given. Important factors which play a role in color vision and notably in color weakness will be discussed, and scientific as well as routine practical tests to study the relationship of these factors will be presented. The relative and combined efficiency of these tests will be explained.

COURSE 221

Room 752 Periods M-2; T-2; W-2

DANIEL B. KIRBY, M.D., New York, N. Y.

A System of Intracapsular Cataract Surgery

The development of a system of intracapsular cataract surgery by which particular technics are adapted to the case in hand will be described. The basis of the system is concerned chiefly with the condition of the lens capsule, cortex, zonule and vitreous. Observations made before and during the progress of the operation guide the surgeon in his selection of the procedure to be employed. An ineffective procedure is not repeated. The application of the system has been made not only to the eyes with cataracts in which the zonule is intact, but also to those in which the lens is luxated or subluxated. The description of procedures applicable to the latter more or less follows the literature, except for the idea of there being necessary the variation of stripping off the resilient, resistant portion of zonule which is present in a case of a young person with congenital malformation.

The methods used in intracapsular extraction in cases with intact zonules represent a further development of the procedures which have been previously described by the author. The cases are divided into three classes: First, a small group of cases in which the zonule is fragile and in which traction or pressure alone or a combination of them may be employed for the relatively easy delivery of the cataract. Second, the large group of cases in which the zonule has the average resiliency and resistance and in which an average degree of pressure, traction and rotation of the lens clockwise and counter clockwise should succeed. The exact method of using these procedures is described. Third, a small percentage of cases in which the zonule exhibits greater than the average degree of resistance and in which when this is recognized, the maneuver of stripping off the zonule is resorted to after the other procedures have been used within the limits of safety. A particular case is described which taught the need for caution. It is recognized that in all cases of cataract formation there is more or less degeneration of the zonule. It is worthwhile to have a procedure which is effective and safe when used properly as proved by experience when others have failed. A report of statistics of results obtained following the procedures described in the system of intracapsular surgery is in the press and will appear shortly. The procedures have been described before, but not in systematic form. It was said before that they were feasible. Now it is reported that they are conservative and desirable.

COURSE 222

Room 756 Periods M-3; T-3; W-3

PETER C. KRONFELD, M.D.
Chicago, Illinois

Gonioscopy — Present Limits

The present status of gonioscopy may be summed up by listing some of its plus as well as its minus values.

Plus Values of Gonioscopy:

1. Gonioscopy offers a simple mechanical explanation for certain glaucomas.
2. Gonioscopy helps in choosing the type of operation most suitable for the case under consideration.
3. Gonioscopy helps in recognizing the causes of failure of glaucoma operations.
4. The information obtained by gonioscopy may be of value in inflammatory, traumatic and neoplastic diseases of the eye.

Minus Values of Gonioscopy:

1. Gonioscopy offers no explanation of the mechanism of certain glaucomas.
2. Gonioscopy requires some special equipment. From 2-4 hours are required to learn the technic of gonioscopy. Considerable experience is required to interpret cases in which the landmarks have been obliterated.
3. Gonioscopy may be conducive to a purely mechanical concept of glaucoma.

COURSE 223

Room 743 Periods T-2; W-2

HEDWIG S. KUHN, M.D., Hammond, Ind.

Examination of Industrial Workers

There will be a discussion of both individual tests and batteries of tests used for screening purposes in cases of industrial applicants and for industrial visual surveys. Outlines will be given of additional tests (close vision, etc.) for special near work jobs; what a visual job-analysis means; refractive problems and

examination for correction for the job and need for increased vigilance in detecting pathology as age level rises.

COURSE 224

Room 720 **Periods M-3; W-3**

RALPH I. LLOYD, M.D., Brooklyn, N. Y.

Macular Lesions

There are certain lesions seen in the fundus which do not cause pain, and microscopic examination of the tissues involved is rarely made. Binocular ophthalmoscopy and red-free light help in the study of some of these cases. At present, it is imperative that the clinical history of these cases be gathered to provide as much information as possible. The interest of the profession is invited to secure microscopic material whenever possible so that, eventually, we may have complete descriptions and correct classification and prognosis.

COURSE 225

Room 776 **Periods T-1; W-1**

PAUL G. MOORE, M.D., Cleveland, Ohio

End Results Following Removal of Foreign Bodies

1. A tabulation and discussion of the results obtained by the author in the removal of intra-ocular foreign bodies.
2. A discussion of the methods used in their removal.
3. When should the anterior or posterior route be used in the removal of a foreign body.
4. The diagnosis, treatment and hospitalization of patients with intra-ocular foreign body.
5. Criticism of technic and results.
6. The removal of superficial foreign bodies and treatment of the resultant wounds.
7. The prognosis as to length of time of disability, time of hospitalization and visual results in eye accidents of this character.
8. The determination of visual acuity.

COURSE 226

Room 702 **Periods M-1; W-1**

W. HOWARD MORRISON, M.D.
Omaha, Nebraska

Malingering

Quick, reliable and easily performed tests for malingering are very important, particularly today. The increasing number of persons employed in hazardous industries and war work carries with it a proportionate increase in injuries and simulated injuries to the visual apparatus. An evaluation of the numerous tests for malingering will be made, and those tests especially suitable for present day practice will be demonstrated.

COURSE 227

Room 754 **Periods T-1; W-2**

RALPH O. RYCHENER, M.D.
Memphis, Tenn.

Common Disorders of the Lids

This discussion will cover the more common and important disorders of the lids which are met in an ophthalmologic practice, most of which can be treated in the office. The diseases are all well illustrated by kodachrome slides, and the discussion pertaining thereto will be of a practical nature.

COURSE 228

Room 778 **Periods T-1; W-1**

GEORGE H. STINE, M.D.
Colorado Springs, Colorado

Differential Diagnosis and Intra-Ocular Localization of Retinal Detachment

Preoperative management of retinal detachment cases, including differential diagnosis and localization of retinal tears, will be discussed in detail. Simple but precise methods of localization will be described, and helpful, practical points outlined. Lantern slides will be shown, and illustrative cases used.

COURSE 229

Room 778 **Periods T-3; W-3**

ARNO E. TOWN, M.D., New York, N. Y.

Contact Lenses

Contact lenses are being recognized more and more as valuable therapeutic agents in keratoconus, corneal dystrophies, keratitis and lid conditions. This course covers the details of the contact glass refraction, the taking of the impressions and the making of the positive from which the glass is constructed.

COURSE 230

Room 740 **Period M-3**

ROBERT VON DER HEYDT, M.D.
Chicago, Ill.

Corneal Dystrophies

1. The M. Buecklers triad.
   1. Crumblike yellow disciform (Fleischer-Fehr type.)
   2. Lattice (Haab-Dimmer type.)
   3. Spotted-later nodular (Groenouw type.)
2. Endothelial and epithelial dystrophies.
   1. Epithelial — a. Secondary to endothelial changes (Fuch's epithelial), b. Increased intra-ocular tension.
3. Vague types — possibly dystrophies.
   1. Dimmer's Keratitis Nummularis.
   2. Salzmann's Keratitis Tuberosa.
   3. Ariboflavinosis.

COURSE 231

Room 740 **Periods T-3; W-3**

HAROLD F. WHALMAN, M.D.
Los Angeles, Calif.

Calibration and Application of the Shahan Thermophore in Ophthalmic Conditions

The Shahan thermophore affords a valuable means of exactly controlled heat application in ophthalmic conditions. The instrument requires calibration, however, to obtain this exactness, as the degree of heat delivered at the applicator tip varies with the size of the tip. A method of calibration will be demonstrated, and the heat necessary in treatment of a variety of conditions will be outlined in accordance with the lecturer's own experience.

COURSE 232

Room 720 **Periods M-1; T-1**

ELMER L. WHITNEY, M.D.
Detroit, Mich.

Dacryocystorhinostomy

The problems of lacrimal disease will be discussed.

The standard surgical methods of dacryocystorhinostomy will be briefly reviewed. Emphasis will be placed on those technics most familiar to the lecturer.

Indications and contraindications for the use of dacryocystorhinostomy will be stressed including the relation to nasal pathology and nasal deformities.

Slides and drawings will be used to illustrate important points in surgical technic and the sources of error in results. A colored movie demonstrating the anastomosis of nasal mucous membrane to tear sac will be shown.

COURSE 233

Room 750 **Periods M-2; W-2**

MEYER WIENER, M.D.
Coronado, Calif.

Plastic Surgery of the Eye

This course will include plastic repair of wounds of the eye and its immediate surroundings, including lids, brow and socket, embracing standard accepted methods as well as lessons learned from experience in the present war. Attention will be called to proper immediate care of injuries of the eye in order to obviate as much as possible the necessity for later major repair work.

COURSE 234

Room 725 **Periods M-1; W-1**

EVERETT H. WOOD, M.D.
Auburn, N. Y.

Interpretation of Central Field Findings

The origin, extent and changes, both pathological and physiological, of angioscotoma will be discussed and illustrated by slides. A demonstration of plotting the angioscotoma will be given.

Individual Courses
OTOLARYNGOLOGY
1943

COURSE 301

Room 710 **Periods: M-1; T-1; W-1**

OSCAR V. BATSON, M.D., Philadelphia

Anatomic Review of Nose

COURSE 302

Room 710 **Periods: M-2; T-2; W-2**

OSCAR V. BATSON, M.D.

Anatomic Review of Pharynx and Larynx

COURSE 303

Room 710 **Periods: M-3; T-3; W-3**

OSCAR V. BATSON, M.D.

Anatomic Review of the Ear

The practical anatomy will be discussed of the several regions indicated, with particular reference to diagnosis, instrumentation and surgical intervention. The illustrative material will be taken from anatomic specimens or from color slides of anatomic specimens. In each one of the subdivisions, the spread of infections from the particular area into adjacent regions will be discussed as well as the invasion of these regions from without. In the discussion of the nose, the commoner variations of the paranasal sinuses will be shown as well as the variations in the nasal fossae and the anatomy of local anesthesia. Concerning the pharynx and larynx, there will be a discussion of the soft palate and a classification of the tracheobronchial tree. In the period on the ear, the essential anatomic features of the several classes of ear operations will be presented as well as the anatomy of the temporal bone and its radiologic identification.

COURSE 304

Room 721 Periods: M-1; W-1

C. D. BLASSINGAME, M.D.
Memphis, Tenn.

The Relationship Between Ludwig's Angina and Other Infections About the Neck

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Pus accumulates most readily in tissues that have the least resistance to pressure. Consequently, the lymphatic arrangement of the mouth and neck, as in other parts of the body, determines the location of primary abscesses and also influences the migratory processes of pus as it seeks to establish itself secondarily. The anatomical arrangement of the mouth and neck favors the development of abscesses in distinct areas. Each of these areas present characteristic factors from the standpoint of diagnosis and treatment. Ludwig's Angina involves only one of these areas, namely, the floor of the mouth, and has its own peculiar clinical aspects. Failure to individualize the symptoms and treatment in this disease accounts, in a large measure, for the high mortality. Abscesses in the submental, lateral pharyngeal, retropharyngeal, pharyngomaxillary and post-visceral spaces each have their characteristics of the lymphatics under the influence of inflammatory reaction and x-radiation and introduce the factor of pressure necrosis which alters the clinical aspects of infections in these areas. The course will include a description of the anatomy of the floor of the mouth and a discussion of the other anatomical spaces about the neck in which abscesses are prone to form. Attention will be given to an evaluation of the various therapeutic measures that are in vogue, viz., surgery, x-radiation, heat and medication.

COURSE 305

Room 736 Periods: M-3; T-3; W-3

LAWRENCE R. BOIES, M.D.
Minneapolis, Minn.

Management of Ear Diseases in Children

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1. Acute otitis media and mastoiditis.
   The present status of our knowledge regarding the effect of the sulfonamides on the incidence of acute otitis media, the management of acute otitis media and mastoiditis with the sulfonamides, the use of the sulfonamides locally, et cetera.
   The modern operation for acute mastoiditis.
2. Chronic suppurative otitis media.
   The importance of an understanding of the etiology and pathogenesis of chronic suppurative otitis media as a basis for therapy.
   Classification.
   Conservative therapy.
   Radical therapy.
   Experiences with the endaural approach.
3. Acquired hearing loss in childhood.
   Inflammation in the eustachian tube and middle ear, the most common cause of acquired hearing loss in childhood.
   The importance of an early recognition of slight to moderate degrees of hearing loss.
   The role of lymphoid tissue.
   Personal experiences with the use of radon in controlling infection in lymphoid tissue.

COURSE 306

Room 718 Periods: M-1; T-1; W-1

JOHN MACKENZIE BROWN, M.D.
Los Angeles, California

Treatment of Acute and Chronic Sinusitis

The treatment of acute pan-sinusitis: General care, local treatment, chemotherapy, treatment of complications. The treatment of subacute pan-sinusitis: Max-

illary, ethmoidal, sphenoidal and frontal. The treatment of acute maxillary sinusitis due to dental infection.

The treatment of chronic sinusitis: General principles of treatment; maxillary sinusitis following nasal infection and from dental pathology; chronic ethmoiditis, sphenoiditis and frontal sinusitis.

The treatment of acute pan-sinusitis: General principles, local therapy, chemotherapy and management of complications. The treatment of the subacute phases.

The treatment of chronic sinusitis: General principles and specific therapeutics of the maxillary, frontal, sphenoidal and ethmoidal sinuses.

The treatment of both acute and chronic maxillary sinusitis following dental infection and extractions, and the therapy for acute and chronic sinusitis in children will be considered.

COURSE 307

Room 718 Periods: M-3; T-3; W-2

PAUL A. CAMPBELL, Lt. Col., M. C. U. S. A., Randolph Field, Texas

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Aviation Otolaryngology

More than 80 per cent of the complaints and discomfort associated with flying have otolaryngological significance. The ear is most vulnerable due to the effects of noise, both audible and inaudible, positive and negative pressure changes due to altitudinal variation and the effects of lessened oxygen supply to the organ of Corti. The sinuses are affected, sometimes to severe degrees if their ventilation is abnormal. The effects of positive and negative pressure changes on mucous membrane lined cavities will be demonstrated. The effect of parachute jumping and power diving will be discussed. Otolaryngological qualifications for the U. S. Army Air Corps will be considered.

COURSE 308

Room 720 Periods: M-2; W-2

THOMAS E. CARMODY, M.D.
Denver, Colo.

Diagnosis and Treatment of Oral Lesions

Examination of lips and mouth.

Complete examination of teeth and gums. This should also include x-rays of teeth and jaws.

X-rays of maxillary sinuses should be studied in connection with those of teeth as infection of these sinuses may be transmitted from or to the teeth and alveolus.

Examination of tongue and floor of mouth including the ducts of the salivary glands.

Palpation both external and internal for enlarged glands and lymph nodes.

X-ray of salivary glands for calculus.

The character of the mucous membrane as to color consistency and secretion.

Careful examination of attachments of tongue to floor of mouth and to posterior structures, even the base, and note made of entrance to pharynx and larynx. This cannot be done without a good light and a laryngeal or mouth mirror.

The palate with its attachments should also be carefully noted.

COURSE 309

Room 725 Periods: M-3; W-3

STUART C. CULLEN, M.D.
Iowa City, Iowa
BY INVITATION

Anesthesia in Otolaryngology

This course will deal with certain fundamentals pertaining to the practical application of anesthetic agents and methods in otolaryngology. An attempt will be made to enumerate a number of simple and effective aids to the successful use of topical infiltration and regional anesthetic methods. Emphasis will be placed on preanesthetic medication and the recognition of and antidated treatment of drug reactions and drug depression. There will be a discussion of all agents and technics available in modern anesthesia with special reference to the endotracheal technic. The advantages and hazards of the rational use of anesthesia by barbiturates introduced intravenously will be cited.

A preliminary report will also be made on the use of curare for the improvement of muscular relaxation during endoscopy.

COURSE 310

Room 726 Periods: M-1; T-1; W-1

DANIEL S. CUNNING, M.D.
New York, N. Y.

The Use of the Sulfonamide Drugs in Otolaryngology

Since the introduction of sulfanilamide, the first of the sulfonamide drugs, in the

treatment of infectious diseases about eight years ago, there have been more than 30 of these drugs placed on the market. At the present time the four most practical drugs of this group that are used more than any in otolaryngology are: Sulfanilamide, sulfapyradine, sulfathiazole and sulfadiazine.

Sulfanilamide apparently is specific for hemolytic Streptococci, sulfapyradine for pneumococci and sulfathiazole for Staphylococci. But sulfadiazine which was brought out in 1940, five years after sulfanilamide, seems to be the least toxic of all the sulfonamide drugs and is the drug of choice today. It is just as efficacious against Streptococci, Staphylococci and pneumococci as the above mentioned drugs that were first heralded as specific against them. Sulfadiazine may be given in the form of sodium sulfadiazine intravenously (50 cc. of a 5% sol.), and the drug shows up in the blood stream within 10 to 15 minutes.

Use of these drugs in ear infections: In the early stages of acute infection of the middle ear as tubotympanic congestion and acute catarrhal otitis media, these drugs are very efficacious and should be given, for in a large percentage of cases it will stop the infection from going any farther. But once the infection passes this stage and has gone on to an acute purulent otitis media, the drum membrane should be opened first, then the drug should be given for a period of 72 hours if necessary, but no longer. If at the end of 72 hours the infection is not better, the drug should be discontinued, for it will probably mask the symptoms and signs of acute mastoiditis. Following mastoidectomy, if the patient is febrile, the drug is indicated; also when complications occur such as sinus thrombosis, brain abscess or meningitis.

In sinusitis: In chronic hyperplastic sinusitis the drug is useless. In cases of purulent sinusitis where the patient is septic, the drug is always indicated especially after the sinuses have been drained. It should also be given when there are complications such as cavernous sinus thrombosis, cellulitis, erysipelas and meningitis.

The drug is always indicated in acute infections of the throat such as tonsillitis, peritonsillar and retropharyngeal abscesses, abscess at the base of the tongue, Ludwig's Angina, laryngotracheal bronchitis, deep infections of the neck, edema of the larynx and erysipelas.

Prophylaxis: It is well to give the drug by mouth for a week before major operations, such as resection of jaw, laryngectomy, purulent pansinusitis cases, etc., as it seems to cut down considerably the infection in the neck, mouth and nose.

Sulfanilamide powder helps a great deal by dusting it into the wound on completion of the operation, as in mastoidectomies, laryngectomies, sinus cases, etc.

These sulfonamide drugs are a valuable adjunct to surgery, but they must not be considered a substitute for surgery. If any surgery is indicated, such as opening an abscess to evacuate pus, or the removal of necrotic bone, this must be done, then the patient should be given large doses of the drug.

The history, mode of action, absorption and excretion therapeutics, toxic manifestations and dosage of the sulfonamides will also be taken up, if time permits.

COURSE 311

Room 739 **Periods: M-3; W-3**

MATTHEW S. ERSNER, M.D.
Philadelphia, Pa.

Chronic Suppurative Otitis Media — Medical and Surgical Treatment

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In the course of chronic suppurative otitis media many pathologic changes take place in various parts of the temporal bone. The mastoid process is most frequently involved and therefore takes the brunt of the infection.

The clinical entities in chronic middle ear suppurations may be classified as: (1) the benign type; (2) the latent type where the discharge is present and is apt to become perilous at any time, and (3) the perilous or dangerous type, where not only may the important structures in the middle ear become involved but those in the temporal region as well.

The diagnosis of the various types may be based on: (1) otorrhea; (2) otitic findings, and (3) the gross symptomatology which depends upon the structures involved.

The treatment in this type of infection may be summed up in two words — medical and/or surgical.

COURSE 312

Room 736 **Periods: M-2; T-1**

FREDERICK A. FIGI, M.D.
Rochester, Minn.

Plastic Surgery in Otolaryngology

Plastic surgery is frequently required in the field of otolaryngology. Deformities and defects of the nose, ears, pharynx and larynx are not uncommon. They may be congenital, acquired or developmental. While their correction is at times optional, often it is essential. Many plastic surgical problems arise in this connection, and a number of procedures of varying usefulness may be employed for restoring function and an approach to normal appearance.

COURSE 313

Room 713 Periods: M-2; T-2; W-2

J. WINSTON FOWLKES, M.D.
New York, N. Y.

Diagnosis and Treatment of Mastoiditis

1. General symptoms of ear disease.
2. Signs and symptoms of mastoiditis.
3. Pathology of acute and chronic mastoiditis.
4. Bacteriology.
5. X-ray findings.
6. Mastoiditis in children as compared with adults.
7. Sulfa therapy.
8. Intracranial complications.
9. Types of operations.
10. Postoperative care.

COURSE 314

Room 705 Periods: M-1; T-1; W-1

A. C. FURSTENBERG, M.D.
Ann Arbor, Mich.

Diseases of the Salivary Glands

An embryological study of the salivary glands reveals anatomical relationships that are exceedingly important to a better understanding of the clinical behavior of disease within these structures. The relation of important nerves and vessels are emphasized and anatomical considerations given to the various parts of the parotid gland that are affected by disease processes. Stress is placed upon the etiological factors in the production of salivary gland disease versus salivary calculi, neighborhood infections, trauma and dehydration after prolonged surgical procedures. Various methods of diagnosis will be discussed and both medical therapy and surgical interference will be reviewed.

COURSE 315

Room 721 Periods: M-3; T-3; W-3

WILLIAM D. GILL, M.D.
San Antonio, Texas

Diseases of the External Ear

A general discussion will be presented of the commoner dermatoses affecting the external ear which are ordinarily classified as otitis extrena. The following clinical types are considered: Cellulitis, acute and chronic; lymphangitis; seborrheic dermatitis-chemical, thermal, mechanical and allergic types; eczema and eczematoid lesions; chemical dermatitis due to iodine, mercury and compounds related to phenol; contact dermatitis and dermatitis venenata; erysipelas; furunculosis; impetigo contagiosa and pyogenic dermatitis; idiosyncrasy; bacterial relationships; Staphylococcus, Streptococcus, B. pyocyaneus; importance of moulds in producing external otitis; classification of fungi commonly encountered; relationship of infection of the external ear to infection in other areas such as the scalp and feet. Methods employed in diagnosis and technic involved. Therapeutic measures: Topical and general; vaccines, fungus extracts and filtrates; trichophytin and bacteriophage. Applicability of radiant energy, ultra-violet rays and x-rays. Sulfanilamide and related compounds. Recurrences and re-infections. Extension to other structures, perichondrium, deeper structures of the ear. Perforation of ear drums. Questions and answers with reciprocal exchange of ideas in discussion.

COURSE 316

Room 739 Periods: T-2; W-2

GLENN J. GREENWOOD, M.D.
Chicago, Ill.

Management of Acute Sinusitis in Children

The incidence of acute sinus disease in children is influenced anatomically by the comparatively large size of the sinus openings, the greater relative approximation of the middle meati to the communicating sinuses and the rich vascularity of these developing structures.

Physiological processes are rapid while the immunity is comparatively low; consequently, the resultant insult is marked, especially when an allergic soil persists or the incidence of acute exanthemata is high.

The management of these cases, particularly when extra or intra-cranial complications occur, emphasizes the importance of adequate early local management supplementing effective general pediatric care.

COURSE 317

Room 705 Periods: M-3; T-3; W-3

FRENCH K. HANSEL, M.D.
St. Louis, Missouri

Allergy of the Nose and Paranasal Sinuses

In the discussion of this subject will be considered the fundamental principles of diagnosis and treatment. A new rapid technic of staining ocular, nasal and bronchial secretions will be demonstrated. Among the various methods of treatment, detailed consideration will be given to dust therapy.

COURSE 318

Room 739 Periods: M-2; T-3

IRVIN HANTMAN, Capt., M. C., U. S. A.
Keesler Field, Miss.

Photography of the Ear Drum Head

1. History of tympanic membrane photography and description of the work of Schulz Van Treeck and Hegener.
2. Discussion of problems involved, both anatomical and technical, and description of apparatus used.
3. Presentation of photographs in natural color of various conditions of the tympanic membrane.
4. Demonstration of technic employed in photographing the drum head (in color) of suitable patients and of an especially prepared skull, with participation by members of the course.

COURSE 319

Room 756 Periods: M-1; W-1

PAUL H. HOLINGER, M.D., Chicago, Ill.

War Injuries of the Larynx

War injuries of the larynx consist of gunshot and shrapnel wounds, fractures, "cut-throat" injuries, stab wounds, inflammatory and destructive changes due to gases and the complications of these specific injuries. The prompt recognition, emergency surgical management and subsequent operative repair of these injuries will be discussed.

COURSE 320

Room 736 Period: W-1

CHEVALIER L. JACKSON, M.D.
Philadelphia, Pa.

Current Trends in the Technic of Bronchoscopy and Endoscopy

Comparative value of different instruments, different modes of anesthesia, different positions of the patient and different methods of use of instruments will be considered. The simplified technic of introduction of the bronchoscope without the preliminary use of the laryngoscope to expose the larynx will be described, and its advantages and disadvantages discussed. Special instruments for the extraction of foreign bodies under fluoroscopic guidance will be demonstrated. The technic of taking tissue for biopsy from bronchial and esophageal lesions will be described, and the relative merits of different types of biopsy forceps will be pointed out. The technic of injection of esophageal varices will be demonstrated.

Finally, one of the most important matters for the bronchologist to study today, namely, the applied anatomy of the tracheobronchial tree, will be described in detail, and its practical value illustrated. In this connection, the indications and technic for the use of the retrograde telescope will be presented.

COURSE 321

Room 752 Periods: M-1; T-1

CHEVALIER L. JACKSON, M.D.
Philadelphia, Pa.

Diagnosis and Treatment of Diseases of Larynx with Particular Reference to Indications and Technic of Direct Laryngoscopy

An attempt will be made to present in this course the anatomy and physiology of the larynx, the means of examining the larynx; then the etiology, pathology, symptomatology and diagnosis, prophylaxis and treatment of laryngeal disease will be discussed. A considerable amount of attention will be given to the differential indirect laryngoscopic diagnosis; then the indications and technic by direct laryngoscopy will be considered. Particular emphasis will be laid on the early

diagnosis of cancer of the larynx and the criteria for the selection of methods of treatment. Finally, the current technic of laryngofissure and laryngectomy will be summarized.

COURSE 322

Room 754 **Periods: M-3; W-3**

W. H. JOHNSTON, M.D.
Santa Barbara, California

Otolaryngologic Therapeutics

This course will consist of a discussion of and an attempt to evaluate the results obtained from the use of therapeutic agents at our disposal for the treatment of diseases of the ear, nose and throat. Local anesthetics and methods of using them will be considered. Conclusions drawn will be based on the experience of a group of active otolaryngologists located in various parts of the country and from the personal experience of the writer.

COURSE 323

Room 743 **Periods: M-1; T-1; W-1**

J. R. LINDSAY, M.D., Chicago, Ill.

Meniere's Symptom Complex — Diagnosis and Treatment

The course includes a review of the routine examination necessary in cases of vertigo, a simple method of recording findings and a brief discussion of the practical significance of the various findings in localizing the site of the lesion and differential diagnosis.

The term Meniere's disease is now commonly used to designate the clinical entity of recurrent vertigo attacks associated with auditory disturbances. The discussion of this disease includes:

1. A simple practical classification into three stages with differential diagnosis in each.
2. Presentation of the characteristic auditory disturbances peculiar to this disease.
3. Demonstration of pathology of the inner ear by lantern slides.
4. Discussion of the medical management and of surgical procedures.

The linking of this clinical condition with a definite pathological entity has provided a more stable basis for therapy.

COURSE 324

Room 776 **Periods: M-2; T-2**

W. J. McNALLY, M.D.
Montreal, Canada

Seasickness and Other Forms of Motion Sickness

In this course will be considered seasickness, air sickness, train sickness, swing sickness, elevator sickness, et cetera. These are disturbances which may follow when the body is subjected to repeated oscillatory movements. They are as old as the first swing or boat.

An attempt will be made to determine what part of the movement is most important in producing motion sickness. The bodily mechanisms involved in the production of motion sickness will be analyzed in order to determine what organs or senses are affected by the stimulus. The part played by the labyrinth will be analyzed and the relative importance of semicircular canal and otolithic stimulation will be dealt with. The matter of individual predisposition, history of susceptibility, acclimatization and habituation will be taken up. An effort will be made to describe the symptomatology. The common remedies will be considered, and the main principles of therapy will be discussed.

COURSE 325

Room 712 **Periods: M-1-2-3; T-1-2-3**

PHILIP E. MELTZER, M.D.
Boston, Mass.

Comparison of the Endaural and External Approach in Mastoid Surgery

Limited to 15 each period

This course will consist of a demonstration of the endaural approach to the regions of the temporal bone which may be involved in ear disease. There will be a discussion of the postauricular approach as compared with the endauricular. Emphasis will be laid on the technic of incision and of instrumentation. Illustrated lecture, discussion and actual demonstration on cadavers will compose the procedure. Dr. Julius Lempert's models will be on view.

COURSE 326

Room 736 Period: M-1
H. P. MOSHER, M.D., Marblehead, Mass.

Osteomyelitis of the Frontal Bone

The discussion will include the histopathology, symptoms, treatment and complications of osteomyelitis of the frontal bone.

COURSE 327

Room 716 Periods: M-1-2; T-1-2; W-1-2
WERNER MUELLER, M.D.
Boston, Mass.

The Diagnosis of Deafness

The most important phase of the problem of deafness is its recognition. In this course the Diagnosis of Deafness will be dealt with under the following headings:

1. History taking.
2. The physical examination.
3. Audiometry.
4. Differential diagnosis.

The subject will be presented entirely from the standpoint of the practicing otologist. Special emphasis is laid on idiopathic deafness.

COURSE 328

Room 754 Periods: M-2; T-2
HORACE NEWHART, M.D.
Minneapolis, Minn.

The Detection and Treatment of Hearing Handicaps Among School Children

The importance of accurate hearing tests of the school population and their value in the prevention of avoidable hearing impairment, the early provision of corrective medical care and educational adjustment will be outlined.

Technics availabe for testing different age groups will be described together with steps toward their standardization and application. The course will deal with essentials of a simple, practical, statewide program, enabling legislation.

COURSE 329

Room 756 Periods: T-2; W-2
SAMUEL J. PEARLMAN, M.D.
Chicago, Ill.

Cellulitis of the Floor of the Mouth

Infections of the floor of the mouth may be severe or mild. The pathology varies from adenitis to periadenitis to cellulitis of the tissues involved. The infection runs its course in preformed potential and actual tissue spaces which communicate one with the other and across the midline. These spaces are:

1. Submental.
2. Sublingual.
3. Submaxillary.
4. The space between the extrinsic muscles of the tongue and parotid spaces.

Complications of infections of the floor of the mouth can be foreseen as logical results of anatomical and pathological necessity, and treatment of these infections can be handled intelligently by an understanding of the anatomy of the areas involved.

COURSE 330

Room 777 Periods: M-1; T-1
CHARLES T. PORTER, M.D.
Boston, Mass.

The Mosher-Toti Tear Sac Operation

A. Historical background of operation.
   1. In 1904 Toti published an account of an external operation on the lachrymal sac.
   2. Distinguishing features between this and the present Mosher-Toti operation.
   3. The evolution of the Mosher-Toti operation with some of the "labor pains" attendant thereon. (The strictly intranasal operation and reasons for its abandonment.)
   4. The development and perfection of the combined intra and extranasal operation.

B. Specimens.
   1. Roentgenograms.
   2. Casts of various stages of the operation with various stages demonstrated by the writer on blackboard.
   3. Demonstration of the actual operation on a wet specimen.

C. Question period.

COURSE 331

Room 724 Periods: M-1; T-1; W-1
ARTHUR W. PROETZ, M.D.
St. Louis, Missouri

Practical Management of the Difficult Sinus Case

The subject will be dealt with under the following headings:

1. Acute ethmoiditis in infants and young children.
2. Persistent purulent pansinusitis in children.
3. Acute frontal sinusitis.
4. Acute maxillary sinusitis.
5. Alveolar maxillary fistula.
6. Sphenoiditis and nasal ganglion pain.
7. Low-grade chronic pansinusitis.
8. Ocular involvements.
9. The over-operated nose.
10. Notes on air conditioning.
11. Notes on climate.
12. The atrophic nose.
13. The allergic nose.
14. The sinuses as foci of infection.

The discussion of these subjects will be limited to the consideration of difficult and unusual cases, as a general presentation of each would occupy more than the allotted time. Standard practices will be taken up only as they apply to such cases. The attitude of the patient toward his difficulty, the feasibility of his carrying out suggested treatment over long periods and the evaluation of therapy will be emphasized.

As the entire list cannot be considered in a given period, the succeeding periods will continue from the point where the previous one terminated.

COURSE 332

Room 706 Periods: M-2; T-2; W-2

JAMES MILTON ROBB, M.D.
Detroit, Michigan

Proven Procedures in Otolaryngological Office Practice

By "proven" is meant those procedures that have been used sufficiently often to obtain good results in an ear, nose and throat practice in the lake district of the middle West over a period of 25 years.

Treatment of diseases of the ear, nose and throat will be taken up separately, their conditions discussed, treatment recommended and the drugs and instruments used to get the best results will be considered. Of special interest will be a presentation of a case of supposed vasomotor, hyperesthetic or allergic rhinitis.

COURSE 333

Room 751 Periods: M-3; T-3; W-3

SAMUEL SALINGER, M.D., Chicago, Ill.

Deformities of the Nasal Septum

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1. A brief summary of the embryology of the septum and how its component parts are developed.
2. Analysis of the "Sphenoid and Huxley Angles" as bearing on the philogenesis of the septum and as pointing to possible reasons for deviations from the midline.
3. Etiology of septum deviations; various theories considered. Question as to the relative importance of developmental factors in contrast to acquired factors such as trauma, etc.
4. Types of septum deviation; ages at which they may be observed and analysis of the location of bends and crests.
5. Surgical correction of septum deformities.
   a. The classic Killian operation.
   b. Freer's modifications.
   c. Metzenbaum's technic.
   d. Variations in technic according to indication.
6. Technic.
   a. Preparation.
   b. Anesthesia.
   c. Instruments.
   d. Closure.
   e. Postoperative dressing.
   f. Postoperative care.
   g. Complications.
7. Septum correction combined with plastic procedures about the nose.
   a. Correction of bony twist.
   b. Removal of hump.
   c. Shortening of the nose.

Illustrations, photographs and models will be provided to clarify many of the points mentioned above.

The classic Killian submucous resection is inapplicable and unsatisfactory in a great many cases because it fails to take into consideration such abnormalities as dislocated septal border, deviations causing external deformity and deviations associated with other nasal abnormalities which are displeasing. As has been pointed out by many observers it is not

always necessary or wise to remove a large amount of cartilage and bone to provide the patient with adequate breathing space. It is just as important to conserve these tissues as far as possible as it is to remove the obstruction. The technic, therefore, should be directed to the attainment of maximum breathing space through minimal sacrifice of supporting structures.

COURSE 334

Room 776 Periods: M-3; W-3

LeROY A. SCHALL, M.D., Boston, Mass.

The Treatment of Malignant Diseases of the Nose and Throat

The nose and nasal accessory sinuses may be the site of either primary or secondary malignant disease. A new growth originating on the eyelid, the lateral nasal wall, superior alveolus or the hard palate may by secondary extension invade either the nasal cavity or the nasal accessory sinuses. Occasionally the nasal accessory sinuses are the site of a metastatic new growth from a distal lesion.

The symptoms suggestive of cancer will vary depending upon the type of lesion and upon the site of the growth.

The treatment will likewise vary as to the variety of the lesion and upon its situation. Superimposed infection will likewise call for appropriate treatment.

COURSE 335

Room 724 Periods: M-3; T-3; W-3

GEORGE E. SHAMBAUGH, JR., M.D.
Chicago, Illinois

Practical Office Diagnosis and Management of Nasal Allergy

The best results in the treatment of chronic nasal allergy will be secured when the allergic factors and the secondary infection so often present are treated simultaneously. It is impractical and uneconomical for the patient with chronic nasal allergy and superimposed infection to make repeated visits to two physicians for his one ailment. The rhinologist should, therefore, be his own allergist.

The methods used in the diagnosis and treatment of chronic nasal allergy will be described and carried out on an illustrative patient, including the taking of a history, cytological examination of the nasal secretions, the technic and interpretation of skin tests, the use of elimination diets and the specific treatment of dust allergy. The results that have been secured in this type of management will be summarized and illustrated by specific case reports.

COURSE 336

Room 731 Periods: M-3; T-3; W-3

JOHN J. SHEA, M.D., Memphis, Tenn.

The Management of the Fractured Bones of the Face

The management of fractures of the bones of the face is similar in principle to that of sinus surgery. As the majority of the fractures extend into one or more of the paranasal sinuses, the most common complication is sinusitis.

The question as to when to correct these fractures in seriously injured patients will be discussed.

The making of suitable appliances for the maintenance of the correction will be demonstrated. The indication and methods of open reduction of the fractures into each of the sinuses will be illustrated.

COURSE 337

Room 743 Periods: M-3; T-3; W-3

W. LIKELY SIMPSON, M.D.
Memphis, Tenn.

The Ethmo-Spheno-Frontal Operation

I. Indications.
II. Preoperative medications.
III. Technic.
  A. Preparation of the skin.
  B. Draping of the patient.
  C. Nasal packing.
  D. General anesthesia.
  F. The incision in the skin and the soft parts.
  G. Retraction sutures.
  H. Hemostasis.
  I. Elevation of the periosteum.
  J. Tying of the anterior ethmoid vessels.
  K. Opening of the bone of the ethmoid and frontal sinuses.
  L. Exenteration of the frontal sinus.
  M. Exenteration of the ethmoid cells.
  N. Procedure for sphenoidectomy.
  O. Closure of the wound.
IV. Complications.

COURSE 338

Room 734 Periods: M-1; T-1; W-1

CLAIRE L. STRAITH, M.D.
Detroit, Michigan
BY INVITATION
WAYNE B. SLAUGHTER, M.D.
Chicago, Illinois
BY INVITATION

Plastic Surgery of Facial Injuries

The treatment of all acutely injured persons must follow a rather definite course, the purpose of which is to give a maximum of benefit to the patient. To obtain this maximum benefit requires adherence to certain fundamental surgical principles, outlined as follows: (Illustrated with kodachrome slides and movies.)

1. Treatment of impending shock or actual shock.
2. Prevention of additional contamination and trauma to the wound. (Use of antiseptics.)
3. Estimation of the patient's general condition and recognition of the presence of multiple injuries.
4. Cleansing of the wound. (Prevention of "tattoo" marks, removal of foreign bodies, etc.).
5. Surgical care. In plastic surgery of the face certain problems are encountered peculiar to this region. These points will be stressed together with emphasis on fixation of fractures, suturing of wounds and reconstructive procedures for recent or old injuries.
6. After care.

COURSE 339

Room 777 Periods: M-3; T-3

O. E. VAN ALYEA, M.D., Chicago, Ill.

Irrigation of Frontal, Maxillary and Sphenoid Sinuses

The ability of the rhinologist to probe the ostia of the frontal, maxillary and sphenoid sinuses is highly important in the treatment of diseases of these structures.

The development of a technic for this procedure is difficult due to anatomic variations of the sinuses and their intranasal communications. Familiarity with these variabilities, however, simplifies the approach to the sinuses, and one equipped with this knowledge should with sufficient application develop a technic which will prove satisfactory in most cases.

Specimens from cadavers will be shown to illustrate the normal anatomic variations. Also, slides and motion pictures will be used to demonstrate cannulization technic.

COURSE 340

Room 725 Periods: M-2; T-2; W-2

FLETCHER D. WOODWARD, M.D.
Charlottesville, Va.

The Management of Recent Faciomaxillary and Mandibular Fractures

The development of high speed transportation has produced an increasing number of such injuries, and now that war is causing so many additional cases, it is necessary that we familiarize ourselves with the many problems presented in the proper management of these wounds. Because of his intimate knowledge of the anatomy of these regions and his surgical training, the responsibility should fall largely on the shoulders of the otolaryngologist.

The subject will be illustrated with numerous lantern slides, and the principles of treatment will be discussed both from the general surgical viewpoint as well as for each particular structure.

The Following Are Condensed Schedules of Courses Previously Described, Arranged for Your Convenience by Days and Periods

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	TITLE	PERIODS USED	Periods Available for Other Subjects
1	**Bedell**	Medical Ophthalmoscopy	3 PERIODS M-3, T-3 and W-3	Six
2	**Dunnington**	Surgery — Eyelids and Orbit	3 PERIODS M-3, T-3 and W-3	Six
3	**Gradle**	Glaucoma	9 PERIODS M-1-2-3, T-1-2-3 and W-1-2-3	None
4	**Hardy**	Orthoptics	6 PERIODS M-2-3, T-2-3 and W-2-3	Three
5	**Hughes**	Surgery of the Lids	2 PERIODS M-2 and T-2	Seven
6	**Lancaster**	Visual Physiology	3 PERIODS M-1, T-1 and W-1	Six
7	**Lyle**	Neuro-Ophthalmology	6 PERIODS M-1-2, T-1-2 and W-1-2	Three
8	**Masters**	Slit Lamp Microscopy	6 PERIODS M-1-2, T-1-2 and W-1-2	Three
9	**Prangen**	Refraction	9 PERIODS M-1-2-3, T-1-2-3 and W-1-2-3	None
10	**Spaeth**	Plastic Repair	6 PERIODS M-2-3, T-2-3 and W-2-3	Three
11	**Theobald**	Pathology	6 PERIODS M-1-2, T-1-2 and W-1-2	Three
12	**White**	Ocular Motor Anomalies	3 PERIODS M-3, T-3 and W-3	Six
13	**White, et al.**	Anomalies of the Extraocular Muscles	6 PERIODS M-1-2, T-1-2 and W-1-2	Three
14	**Woods**	Ocular Syphilis	2 PERIODS M-2 and T-2	Seven

CONTINUOUS COURSES — OTOLARYNGOLOGY

51	**Clerf**	Hoarseness	3 PERIODS M-2, T-2 and W-2	Six
52	**Hughson**	Hearing Aids	3 PERIODS M-3, T-3 and W-3	Six
53	**Jones**	Causes of Dizziness	2 PERIODS M-1 and T-1	Seven
54	**Kazanjian**	War Injuries — Face and Jaws	3 PERIODS M-3, T-3 and W-3	Six
55	**Kelly**	Bilateral Paralysis — Abductor Muscles	3 PERIODS M-1, T-1 and W-1	Six
56	**Kopetzky**	What Can Be Done for the Deafened	3 PERIODS M-3, T-3 and W-3	Six
57	**Reger**	Audiometers and Hearing Aids	2 PERIODS M-2 and T-2	Seven
58	**Sellers**	Equilibrium and Vertigo	2 PERIODS M-3 and W-3	Seven

SCHEDULE OF INDIVIDUAL COURSES

Key: M-1 1st Period: 8:30 a.m. to 9:30 a.m.

MONDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
205	Beach	Refraction
209	Burch	Retinal Detachment — End Results
213	Crisp	Cross Cylinders
217	Frost	External Diseases — Eye
220	Glasser	Color Vision
226	Morrison	Malingering
232	Whitney	Dacryocystorhinostomy
234	Wood	Central Field Findings

Key: M-1 1st Period: 8:30 a.m. to 9:30 a.m.

MONDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
301	Batson	Nose — Anatomic Review
304	Blassingame	Ludwig's Angina
306	Brown	Sinusitis
310	Cunning	Sulfanomide Drugs
314	Furstenberg	Salivary Glands
319	Holinger	War Injuries — Larynx
321	Jackson	Larynx
323	Lindsay	Meniere's Symptoms Complex
325	Meltzer	Mastoid Surgery
326	Mosher	Osteomyelitis — Frontal Bone
327	Mueller	Deafness — Diagnosis
330	Porter	Toto-Mosher Tear Sac Operation
331	Proetz	Difficult Sinus Cases
338	Straith and Slaughter	Plastic Surgery

Key: M-2 2nd Period 10 a.m. to 11 a.m.

MONDAY

Ophthalmology

No. Course	SPEAKER	SUBJECT
201	Adson	Neuralgias of the Face
203	Atkinson	Anesthesia — Ophth.
204	Bahn	Visual Requirements — Armed Services
212	Cowan	Presbyopia
214	Davis	Eye Defects — School Child
218	Gibson	Strabismus
221	Kirby	Intracapsular Cataract Extraction
233	Wiener	Plastic Surgery — Eye

Key: M-2 2nd Period 10 a.m. to 11 a.m.

MONDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
302	Batson	Pharynx and Larynx
308	Carmody	Oral Lesions
312	Figi	Plastic Surgery — Oto.
313	Fowlkes	Mastoiditis
318	Hantman	Ear Drum-head Photography
324	McNally	Seasickness
325	Meltzer	Mastoid Surgery
327	Mueller	Deafness — Diagnosis
328	Newhart	Hearing Handicaps — School Children
332	Robb	Office Practice — Oto.
340	Woodward	Recent Faciomaxillary and Mandibular Fractures

Key: M-3 3rd Period: 11:30 a.m. to 12:30

MONDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
206	Berens	Uveitis
207	Boeder	Aniseikonia
215	Falls	Hereditary Aspects — Oph.
216	Frey	Office Practice — Oph.
222	Kronfeld	Gonioscopy
224	Lloyd	Macular Lesions
230	Von der Heydt	Corneal Dystrophies

Key: M-3 3rd Period: 11:30 a.m. to 12:30

MONDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
303	Batson	Ear — Anatomic Review
305	Boies	Ear Diseases — Children
307	Campbell	Aviation — Oto.
309	Cullen	Anesthesia — Oto.
311	Ersner	Chronic Suppurative Otitis Media
315	Gill	External Ear — Diseases
317	Hansel	Nose and Paranasal Sinuses
322	Johnston	Therapeutics — Oto.
325	Meltzer	Mastoid Surgery
333	Salinger	Nasal Septum — Deformities
334	Schall	Malignant Diseases — Nose and Sinuses
335	Shambaugh	Nasal Allergy
336	Shea	Fractures — Bones of the Face
337	Simpson	Ethmo-Spheno Frontal Operation
339	Van Alyea	Sinuses — Irrigation

Key: T-1 1st Period: 8:30 a.m. to 9:30 a.m.

TUESDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
202	Alvis	Acute Corneal Lesions
209	Burch	Retinal Detachment — End Results
210	Carroll	Optic Neuritis
217	Frost	External Diseases — Eye
220	Glaser	Color Vision
225	Moore	Foreign Bodies
227	Rychener	Diseases of the Lids
228	Stine	Retinal Detachment
232	Whitney	Dacryocystorhinostomy

Key: T-1 1st Period: 8:30 a.m. to 9:30 a.m.

TUESDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
301	Batson	Nose — Anatomic Review
306	Brown	Sinusitis
310	Cunning	Sulfanomide Drugs
312	Figi	Plastic Surgery
314	Furstenberg	Salivary Glands
321	Jackson	Larynx
323	Lindsay	Meniere's Symptoms Complex
325	Meltzer	Mastoid Surgery
327	Mueller	Deafness — Diagnosis
330	Porter	Toto-Mosher Tear Sac Operation
331	Proetz	Difficult Sinus Cases
338	Straith and Slaughter	Plastic Surgery

Key: T-2 2nd Period: 10 a.m. to 11 a.m.

TUESDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
201	Adson	Neuralgias of the Face
203	Atkinson	Anesthetics — Ophth.
204	Bahn	Visual Requirements — Armed Services
208	Brown	Ocular Tuberculosis
212	Cowan	Presbyopia
214	Davis	Eye Defects — School Child
219	Gillette	Vitreous Degeneration
221	Kirby	Intracapsular Cataract Extraction
223	Kuhn	Examination of Industrial Workers

Key: T-2 2nd Period: 10 a.m. to 11 a.m.

TUESDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
302	Batson	Pharynx and Larynx
313	Fowlkes	Mastoiditis
316	Greenwood	Acute Sinusitis in Children
324	McNally	Seasickness
325	Meltzer	Mastoid Surgery
327	Mueller	Deafness — Diagnosis
328	Newhart	Hearing Handicaps — School Children
329	Pearlman	Cellulitis
332	Robb	Office Practice — Oto.
340	Woodward	Recent Faciomaxillary and Mandibular Fractures

Key: T-3 3rd Period: 11:30 a.m. to 12:30

TUESDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
206	Berens	Uveitis
207	Boeder	Aniseikonia
211	Castroviejo	Corneal Plastic Surgery
215	Falls	Hereditary Aspects — Oph.
216	Frey	Office Practice — Oph.
222	Kronfeld	Gonioscopy
229	Town	Contact Lens
231	Whalman	Shahan Thermophore

Key: T- 3 3rd Period: 11:30 a.m. to 12:30

TUESDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
303	Batson	Ear — Anatomic Review
305	Boies	Ear Diseases — Children
307	Campbell	Aviation — Oto.
315	Gill	External Ear — Diseases
317	Hansel	Nose and Paranasal Sinuses
318	Hantman	Ear Drum-head Photography
325	Meltzer	Mastoid Surgery
333	Salinger	Nasal Septum — Deformities
335	Shambaugh	Nasal Allergy
336	Shea	Fractures — Bones of the Face
337	Simpson	Ethmo-Spheno Frontal Operation
339	Van Alyea	Sinuses — Irrigation

Key: W-1 1st Period: 8:30 a.m. to 9:30 a.m.

WEDNESDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
202	Alvis	Acute Corneal Lesions
210	Carroll	Optic Neuritis
211	Castroviejo	Corneal Plastic Surgery
213	Crisp	Cross Cylinders
219	Gillette	Vitreous Degeneration
225	Moore	Foreign Bodies
226	Morrison	Malingering
228	Stine	Retinal Detachment
234	Wood	Central Field Findings

Key: W-1 1st Period: 8:30 a.m. to 9:30 a.m.

WEDNESDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
301	Batson	Nose — Anatomic Review
304	Blassingame	Ludwig's Angina
306	Brown	Sinusitis
310	Cunning	Sulfanomide Drugs
314	Furstenberg	Salivary Glands
319	Holinger	War Injuries — Larynx
320	Jackson	Bronchoscopy and Endoscopy
323	Lindsay	Meniere's Symptoms Complex
327	Mueller	Deafness — Diagnosis
331	Proetz	Difficult Sinus Cases
338	Straith and Slaughter	Plastic Surgery

Key: W-2 2nd Period: 10 a.m. to 11 a.m.

WEDNESDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
208	Brown	Ocular Tuberculosis
218	Gibson	Strabismus
221	Kirby	Intracapsular Cataract Extraction
223	Kuhn	Examination of Industrial Workers
227	Rychener	Diseases of the Lids
233	Wiener	Plastic Surgery — Eye

Key: W-2 2nd Period 10 a.m. to 11 a.m.

WEDNESDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
302	Batson	Pharynx and Larynx
307	Campbell	Aviation — Oto.
308	Carmody	Oral Lesions
313	Fowlkes	Mastoiditis
316	Greenwood	Acute Sinusitis in Children
327	Mueller	Deafness — Diagnosis
329	Pearlman	Cellulitis
332	Robb	Office Practice — Oto.
340	Woodward	Recent Faciomaxillary and Mandibular Fractures

Key: W-3 3rd Period: 11:30 a.m. to 12:30

WEDNESDAY

Ophthalmology

Course No.	SPEAKER	SUBJECT
207	Boeder	Aniseikonia
222	Kronfeld	Gonioscopy
224	Lloyd	Macular Lesions
229	Town	Contact Lens
231	Whalman	Shahan Thermophore

Key: W-3 3rd Period: 11:30 a.m. to 12:30

WEDNESDAY

Otolaryngology

Course No.	SPEAKER	SUBJECT
303	Batson	Ear — Anatomic Review
305	Boies	Ear Diseases — Children
309	Cullen	Anesthesia — Oto.
311	Ersner	Chronic Suppurative Otitis Media
315	Gill	External Ear — Diseases
317	Hansel	Nose and Paranasal Sinuses
322	Johnston	Therapeutics — Oto.
333	Salinger	Nasal Septum — Deformities
334	Schall	Malignant Diseases — Nose and Sinuses
335	Shambaugh	Nasal Allergy
336	Shea	Fractures — Bones of the Face
337	Simpson	Ethmo-Spheno Frontal Operation

Trial Notes

It may be helpful in orienting selections, if you use this trial schedule prior to filling in the order sheet.

MORNING HOURS

	M (Monday)	T (Tuesday)	W (Wednesday)
1st Period	Course No.		
	Instructor		
2nd Period	Course No.		
	Instructor		
3rd Period	Course No.		
	Instructor		

BUSINESS MEETING

Crystal Room — 5:30 P.M.

TUESDAY, OCTOBER 12

Reports of Standing Committees will be read and later published in the November-December issue of the TRANSACTIONS. The Senior Member of the Council will report Council action upon Committee recommendations because changes in committee reports as recommended by the Council, or new business originating from the floor, must be referred to the Council for action.

ORDER OF BUSINESS

1—Call to Order by the President
2—Report of the Executive Secretary-Treasurer
3—Report of Historian
4—Report of Senior Member of Council
5—Representatives on Joint Committees
6—Discussion of Reports by Members
7—Unfinished Business
8—New Business
9—Changes in Membership Classification
  Honorary Members
  Life Members
  Senior Members

1943 Candidates

Objections to the election of a candidate must be filed with the Executive Secretary previous to **Sunday, October 10, 1943.**

CANDIDATES CERTIFICATED BY BOARDS

Ophthalmology (Op.)
Otolaryngology (ALR.)
Plastic Surgery (Pl.S.)

Adix, H. Victor Jr., Lt. Comdr. (MC) U. S. Naval Hospital, Bremerton, Washington, ALR.

Allen, Wilmer L., Capt. (MC) Station Hospital, Camp Ellis, Ill., ALR.

Bair, Hugo L., Mayo Clinic, Rochester, Minn., Op.

Baldwin, Thomas M., 222 Brodhead Bldg., Beaver Falls, Pa., ALR.

Barrett, John A., 1304 Walker Ave., Houston, Texas, ALR.

Beil, Wallace C., 812 Maison Blanche Bldg., New Orleans 16, La., Op.

Berman, Harry L., 1142 Chapel, New Haven 11, Conn., ALR.

Berney, Irving Vincent, 31 Lincoln Park, Newark 2, N. J., ALR.

Bisno, Daniel, 4500 Olive St., St. Louis 8, Missouri, Op.

Blaydes, J. E., First Nat'l Bk. Bldg., Bluefield, W. Va., Op.

Bloch, Charles L., Lt. (MC) USNR Post Dispensary, Quantico, Va., Op.

Blumklotz, Bruno, 185 N. Wabash Ave., Chicago 1, Ill., ALR.

Bodenheimer, Ernst, 1212 Eutaw Pl., Baltimore 17, Md., Op.

Bower, Richard L., 1103 Grand Ave., Kansas City 6, Missouri, ALR.

Brady, Charles Henry, Sedalia, Mo., ALR.

Brooks, Clyde Montgomery, Capt. (MC) 105 S. Norman Ave., Carbondale, Ill., ALR.

Brooks, Jack E., 1825 West Harrison, Chicago 12, Ill., ALR.

Brown, Henry A., Mayo Clinic, Rochester, Minn., ALR.

Brown, Wilbur Max, Major (MC) 28½ W. 4th St., Mansfield, Ohio, Op.

Bryan, James H., 508 N. Grand Blvd., St. Louis 3, Missouri, Op.

Buchbinder, Murray W., Major (MC), 113th Infantry Armory, Sussex Avenue and Jay St., Newark, N. J., ALR.

Budetti, Joseph A., Lt. (MC) 90 Sherman Ave., Mt. Vernon, N. Y., ALR.

Burston, Herschel, 1917 Wilshire Blvd., Los Angeles 5, Calif., ALR.

Cammisa, James J. Vincent, Comdr. (MC) U. S. Navy Yard Dispensary, Washington 25, D. C., ALR.

Cannon, Harold J., 536 W. Wisconsin Ave., Milwaukee 3, Wis., ALR.

Cetner, John A., 248 State St., Albany 6, N. Y., Op.

Chapman, E. J., 331 Haywood Bldg., Asheville, N. C., ALR.

Clapp, C. A., 513 N. Charles St., Baltimore 1, Md., Op.

Clark, Williard C., 530 Fidelity Bldg., Dayton 2, Ohio, Op.

Clothier, William L., 407-411 Kane Bldg., Pocatello, Idaho, Op.
Conley, John J., Capt. (MC) 313 Rowland Ave., Carnegie, Pa., ALR.
Constantine, Elizabeth Frederick, 927 Park Ave., New York 28, N. Y., Op.
Cooper, James H., University Hospital, Ann Arbor, Mich., Op.
Cotten, Willis Frank, 202 State Street, McComb, Miss., ALR.
Cranston, William J., Jr. Capt. (MC), 175 Clinton Ave., Kingston, N. Y., Op.
Crawford, H. E., 139 Kinoole St., Hilo, Hawaii, T. H., Op.
Cunningham, Bernard Poland, Capt. (MC) Harvard Unit, Medical School, Boston 15, Mass., ALR.
Cunningham, John C., Capt. (MC), 1200 Main St., Dubuque, Iowa, Op.
Cusick, Paul L., Capt. (MC), Mayo Clinic, Rochester, Minn., Op.
Damitz, John Cyril, (MC) USNR, 1036 Emma Ave., Akron 2, Ohio, OALR.
Daniel, Ruby K., Medical Arts Bldg., Dallas 1, Texas, Op.
Deems, Myers B., Capt. (MC) Fitzsimons General Hospital, Denver, Colorado ALR.
Dolowitz, David A., Johns Hopkins Hospital, Baltimore, Md., ALR.
Donlon, John J., 21 S. Village Ave., Rockville Center, N. Y., ALR.
Donovan, Richard Joseph, 1400 Hancock St., Quincy, Mass., OALR.
Downing, Arthur H., Lt. (MC) Medical Induction Station, Fort Snelling, Minn., Op.
Drews, Leslie C., 516 Metropolitan Bldg., St. Louis 3, Missouri, Op.
Dubins, Henry B., 39-15 Main Street, Flushing, N. Y., Op.
Elliot, Alfred J., Night Vision Laboratory, Physics Bldg., McGill University, Montreal, Canada, Op.
Faggart, George Henry, 18 W. Oglethorpe Ave., Savannah, Georgia, ALR.
Faier, Herman I., Major (MC) Hoff General Hospital, Santa Barbara, Calif., Op.
Falls, Harold Francis, University Hospital, Ann Arbor, Mich., Op.
Fitzgerald, J. R., 30 N. Michigan Ave., Chicago 2, Ill., Op.
Follweiler, Frank L., 423 York Road, Jenkintown, Pa., ALR.
Foote, William D., Lt. (MC) Station Hospital, Hunter Field, Savannah, Ga., Op.
Forrester, Alex M., Roswell Army Air Field, Station Hospital, Roswell, New Mexico, ALR.
Fowler, Floyd J., Akron Clinic, 513 W. Market St., Akron 3, Ohio, ALR.
Fox, Samuel L., 1820 Eutaw Place, Baltimore 17, Md., ALR.
Frank, Herman W., Medical Bldg., Gadsden, Alabama, Op.
Frey, Walter D., 200 N. Upper St., Lexington 15, Ky., Op.
Gans, Jerome A., 950 East 59th St., Chicago 37, Ill., Op.
Gibson, William J., 650 Main St., New Rochelle, N. Y., ALR.
Gillis, Marion Howard, Jr., Salisbury, Md., Op.
Gissy, C. J., 717 Metropolitan Bldg., St. Louis 3, Missouri, Op.
Goldmacher, H. B., 113 Elmora Ave., Elizabeth 2, N. J., ALR.
Goodwin, Vernon L., 509 Central Tower, Youngstown, Ohio, ALR.
Griesemer, Z. Lawrence, 1145 E. Jersey St., Elizabeth 4, N.J., Op.
Guzek, Joseph T., 29 Greene Ave., Brooklyn 5, N. Y., Op.
Hagood, Daniel S., 822 First Nat'l Bk. Bldg., Montgomery 4, Ala., Op.
Haik, George M., Capt. (MC) 812 Maison Blanche, New Orleans 16, La., Op.
Haines, Henry L., Johns Hopkins Hospital, Baltimore, Md., ALR.
Hanser, Samuel Albert, Major (MC) 3109 Allen Ave., St. Louis 4, Missouri, Op.
Hauser, Maurice J., Major (MC) Station Hosp., Fort Custer, Mich., Op.
Hawkins, N. W., 109 West Liberty St., Farmington, Missouri, ALR.
Hearn, Paul P., Capt. (MC) Station Hosp., Tyndall Field, Panama City, Fla., ALR.
Hedemark, Norman G., Capt. (MC) 518 Eastman Bldg., Boise, Idaho, ALR.
Henry, L. L., 595 E. Colorado St., Pasadena 1, Calif., Op.
Hillyer, Ernest C., Capt. (MC) 217 Bennett Street, Long Beach 3, Calif., Op.
Hoeltgen, Maurice M., 1607 West 51st St., Chicago 9, Ill., Op.
Hoffman, N. Fulmer, Jr., 2035 Delancey Pl., Philadelphia 3, Pa., ALR.
Holmen, Robert W., 1051 Lowry Medical Arts Bldg., St. Paul 2, Minn., ALR.
Holt, Helen, 720 North Michigan Ave., Chicago 11, Ill., Op.
Howard, H. Sargent, 418 Jefferson Bldg., Peoria 2, Ill., ALR.
Hoxie, Derrick, Bldg. 109, U.S.N.T.S. Great Lakes, Ill., ALR.
Hughes, William F. Jr., Johns Hopkins Hospital, Baltimore 5, Md., Op.
Hurwitz, Paul, Major (MC) Station Hosp., Camp Ellis, Ill., Op.
Isbell, Euclid A., 948 Forrest Ave., Gladsden, Ala., ALR.

Jarrott, James T., 131 Fulton Ave., Hempstead, N. Y., Op.
Jerome, Bourne, Major (MC) Station Hosp., Fort Sheridan, Ill., Op.
Jones, W. Yerby, 436 Sycamore St., Buffalo 4, N. Y., Op.
Jordan, Philip J., 910 Medico-Dental Bldg., San Jose 20, Calif., ALR.
Kennedy, Francis L., Missouri Theatre Bldg., St. Louis 3, Missouri, ALR.
Kershner, C. M., Watson Clinic, Brookings, S. D., Op.
Kimata, Harold T., Lt. (MC) Station Hosp. No. 2, Fort Bragg, N. C., ALR.
Kirch, Walter, 417 Bankers Trust Bldg., Des Moines 9, Iowa, ALR.
Klawans, Harold L., Capt. (MC) McCloskey General Hosp., Temple, Texas, ALR.
Koch, Harold J., 6235 S. Kedzie Ave., Chicago 29, Ill., Op.
Kos, Clair M., Capt. (MC) School of Aviation Med., Randolph Field, Texas, ALR.
Krieger, Alexander A., Major (MC) 2145 Wightman St., Pittsburgh 17, Pa. Op.
Lee, Wayne Robert, 613 Medical Arts Bldg., Burlington, Iowa, ALR.
Lempert, Julius, 119 East 74th St., New York 21, N. Y., ALR.
Leopold, Irving H., University Hospital, University of Pa., Philadelphia 4, Pa., Op.
Lewis, Samuel D., 49 Pearl St., Hartford 3, Conn., Op.
Lippmann, Otto, 406 Scarborough Bldg., Austin 22, Texas, Op.
Loder, L. L., 506 Hackley Union Nat'l Bk. Bldg., Muskegon, Mich., Op.
Longaker, Edwin P., 41 E. Montgomery Ave., Ardmore, Pa., ALR.
Loomis, George L., 310 Choate Bldg., Winona, Minn., ALR.
Loy, A. W., Comdr. (MC) Sch. of Aviation Med., U. S. Naval Air Station, Pensacola, Fla., Op.
McCall, J. W., 610 Rose Bldg., Cleveland 15, Ohio, ALR.
McGarry, H. Isabelle, 636 Church St., Evanston, Ill., Op.
McGowan, Joseph Francis, New Medical Bldg., Asheville, N. C., ALR.
McGuire, John, Peery Bldg., Bluefield, W. Va., ALR.
McKay, Edward D., Capt. (MC) 5th and Hazel, Texarkana, Arkansas-Texas, Op.
McLaughlin, R. C., 3780 Wilshire Blvd., Los Angeles 5, Calif., ALR.
McMurray, John S., 636 Washington Trust Bldg., Washington, Pa., ALR.
MacKenzie, J. Grant, Fl. Lt. RCAF (MC) 50 Gould St., Toronto, Ont., Can., ALR.
Macpherson, William A., Capt. (MC) 2701 East Florence Ave., Huntington Park, Calif., Op.
Mahoney, Frank A. Jr., Capt. (MC) 16 Harriet St., Springfield 7, Mass., ALR.
Maier, Marcus M., 513 Front St., Hempstead, N. Y., ALR.
Mann, Mortimer, 323 Hume-Mansur Bldg., Indianapolis 4, Ind., Op.
Marmelstein, Maurice, First Nat'l Bk. Bldg., Carbondale, Pa., Op.
Master, John R., Lt. (MC) Letterman General Hosp., San Francisco, Calif., ALR.
Matthews, John L., Major (MC) Sch. of Aviation Med., Randolph Field, Texas, Op.
Matzkin, Lloyd L., Capt. (MC) Station Hosp., Ft. Jackson, Columbia, S. C., ALR.
Maxwell, Hal W., Lt. (MC) Myra, Texas, Op.
Merz, Earl H., 55 E. Washington St., Chicago 2, Ill., Op.
Milder, Benjamin, 501 Humboldt Bldg., St. Louis 3, Missouri, Op.
Miller, A. Boyd, 1006 Professional Bldg., 429 Penn Ave., Pittsburgh, Pa., ALR.
Miller, Sidney N., 53 S. Hamilton St. Poughkeepsie, N. Y., ALR.
Minsky, Arthur, 2 E. 95th St., New York 28, N. Y., Op.
Mohun, Meade, 23 Second Ave., San Mateo, Calif., ALR.
Monaco, Louis, 523 West Sixth St., Los Angeles 14, Calif., ALR.
Moore, Donald E., 1100 Euclid Ave., Syracuse 10, N. Y., Op.
Morrison, James D., Capt. (MC) 208 North 28th St., Billings, Mont., Op.
Morrone, Francis X., Brooklyn Eye and Ear Hosp., 29 Greene Ave., Brooklyn 5, N. Y., ALR.
Moser, George P., Capt. (MC) Bloomsburg, Pa., ALR.
Newell, Frank W., Lt. (MC), 108 Gen. Hosp., New Orleans, La., Op.
Newton, Ray E., 1010 Reynolds Bldg., Jackson, Mich., Op.
Norris, Mary Alice, 404 Hume-Mansur Bldg., Indianapolis 4, Ind., Op.
Overton, Jesse W., 324 East 41st St., New York 17, N. Y., ALR.
Owen, Walter E., 942 Jefferson Bldg., Peoria 2, Ill., ALR.
Patch, Orien B., Capt. (MC) U. S. A. A. F. T. T. C., Station Hosp., Lincoln Air Base, Lincoln 1, Neb., ALR.
Paton, Walter M., 919 Medical-Dental Bldg., Vancouver, B. C., Can., ALR.
Pattee, George L., 612 Republic Bldg., Denver 2, Colo., ALR.
Pearson, George J., 320 N .Third, Burlington, Iowa, ALR.

Penta, John J., 313 Washington, Reading, Pa., ALR.
Peter, Beverly K., Beckley Hospital, Beckley, W. Va., ALR.
Phillips, James R., Major (MC) Station Hosp., Camp Livingston, La., Op.
Pierce, George W., 490 Post St., San Francisco 2, Calif., PLS.
Platt, Edward S., Lt. Comdr. (MC) 4 Hamilton Ave., Auburn, N. Y., Op.
Portnoy, Samuel H., 19 Garfield Place, Cincinnati 2, Ohio, ALR.
Presnell, C. A., 634 N. Grand Ave., St. Louis 3, Missouri, ALR.
Pugmire, Ralph W., 2404 Washington Blvd., Ogden, Utah, Op.
Qualls, H. W., 1635 Exchange Bldg., Memphis 3, Tenn., ALR.
Quinlan, Joseph D., 44 Prichard St., Fitchburg, Mass., ALR.
Raines, Morris M., 34 N. Ash St., Ventura, Calif., ALR.
Ravin, Louis C., 316 Michigan St., Toledo 2, Ohio, Op.
Riter, Kersey Clyde, Capt. (MC) 3 N. Main St., Logan, Utah, ALR.
Robertson, E. Norris, Jr., 508 N. Grand, St. Louis 3, Missouri, Op.
Robison, J. M., 1304 Walker Ave., Houston, Texas, ALR.
Ronis, Bernard J., Capt. (MC) 7141 Elmwood Ave., Philadelphia 42, Pa., ALR.
Rosen, David M., 19 West 55th St., New York 19, N. Y., ALR.
Rosen, Emanuel, Capt. (MC) 692 High St., Newark 2, N. J., Op.
Rothenberger, Marvin K., 206 N. 8th St., Allentown, Pa., ALR.
Royer, Charles A., Lt. (MC) 431 College Ave., Alva, Okla., Op.
Rubin, Henry J., 611 N. Stanley Ave., Los Angeles, Calif., ALR.
Rubin, Herman G., Major (MC) Station Hosp., Fort Sill, Okla., ALR.
Ryder, William H., 185 Church St., New Haven, Conn., Op.
Sacheroff, Louis, 422 Keith Bldg., Cleveland, Ohio, ALR.
Sadoff, Irvin E., Lt. (MC) 1229th Reception Center, Fort Dix, N. J., ALR.
Samponaro, Nicholas, 650 Main St., Hartford 3, Conn., ALR.
Sauer, John J. 30 East 40th St., New York 16, N. Y., Op.
Scheffler, Milton Morris, 58 E. Washington St., Chicago 2, Ill., Op.
Scholz, Roy O., Johns Hopkins Hospital, Baltimore 5, Md., Op.
Schwarz, Gerald T., 10515 Carnegie Ave., Cleveland 6, Ohio, Op.
Scobee, R. G., 640 S. Kingshighway, St. Louis 10, Missouri, Op.
Scurti, Bernard V., 51 Harmon St., Brooklyn 21, N. Y., ALR.
Seip, Walter R., Capt. (MC) 429 Penn Ave., Pittsburgh 22, Pa. Op.
Shanedling, P. D., 310 S. Michigan Ave., Chicago 4, Ill., Op.
Simmons, J. Thomas, Lt. (MC) 316 W. 10th St., Erie, Pa., Op.
Sitney, Julian J., Lt. (MC) 58 E. Washington, Chicago, Ill., Op.
Smith, Leslie M., U. S. Marine Hosp., Baltimore 11, Md., Op.
Smith, Norman N., 291 Whitney Ave., New Haven 11, Conn., ALR.
Smith, Thomas T., Jefferson Hosp., Philadelphia 7, Pa., ALR.
Stauffer, Harry B., 234 Madison, Jefferson City, Missouri, ALR.
Stevenson, Walter D., Jr., W. C. U. Bldg., Quincy, Ill., ALR.
Stewart, Charles E., Capt. (MC) Percy Jones Gen. Hosp., Battle Creek, Mich., ALR.
Stoner, Donald D., 156 W. Pomfret St., Carlisle, Pa., ALR.
Strause, Harold L., 826 N. 5th St., Reading, Pa., ALR.
Strauss, Arthur, 130 Pavilion Ave., Long Branch, N. J., ALR.
Strickler, Alfred D., 26 N. 9th St., Lebanon, Pa., Op.
Stuard, Charles G., Major (MC) Medical Arts Bldg., Tulsa 3, Okla., Op.
Swan., Kenneth C., University Hosp., Iowa City, Iowa, Op.
Tangen, George M., University of Minnesota, Minneapolis 14, Minn., ALR.
Thacker, Edgar A., 602 W. University Ave., Urbana, Ill., ALR.
Thomas, Charles I., 10515 Carnegie Ave., Cleveland 6, Ohio, Op.
Thompson, William R., Major (MC) 281 S. Ashland Ave., Lexington 37, Ky., ALR.
Tinkess, Donald E., Major (MC) Ituri Towers, Greenwich, Conn., Op.
Tyler, Richard E., The Norwood Clinic, 2501 N. 16th Ave., Birmingham 4, Ala., ALR.
Van Wien, Stefan, Lt. (MC) 104 S. Michigan Blvd., Chicago, Ill., Op.
Weaver, Emerson M. F., Major (MC) Sch. of Aviation Med., Randolph Field, Texas, Op.
Webb, M. A., 4500 Olive St., St. Louis, Mo., ALR.
Wellford, B. R., 510 Medical Arts Bldg., Richmond 19, Va., ALR.
Welt, Bernard, 135 New York Ave., Brooklyn, N. Y., ALR.

Wesson, Thomas W., 124 S. E. First, Evansville 8, Ind., ALR.
Wiest, Philip R., Capt. (MC) 1036 Franklin St., Reading, Pa., ALR.
Wilcox, Malcolm B., 5 West 21st St., Kearney, Neb., ALR.
Williams, John W., Lt. (MC) Medical Arts Bldg., Springfield, Missouri, ALR.
Williams, Russell I., 422 Hynds Bldg., Cheyenne, Wyo., ALR.
Wilmes, Malcolm N., Major (MC) Station Hosp., Camp Adair, Oregon, ALR.
Winer, Albert S., 1835 Eye St., N. W., Washington, D. C., ALR.
Wojniak, Frank, 4649 S. Ashland, Chicago 9, Ill., ALR.
Wolf, Lewis R., 255 S. 17th St., Philadelphia 3, Pa., Op.
Woolfolk, William L., 115 E. 4th St., Owensboro, Ky., ALR.
Work, Walter P., 384 Post St., San Francisco, Calif., ALR.
Zeman, Michael S., Capt. (MC) Station Hospital, Camp Phillips, Kansas, ALR.

CANDIDATES NOT CERTIFICATED

(Eligible for Membership After Passing Board Examinations)

Byrnes, Victor A., School of Aviation Medicine, Randolph Field, Texas
Carrozza, Natale S., 108 Vanderbilt St., Brooklyn 18, N. Y.
Collins, John A., 406-407 Union Savings and Trust Bldg., Warren, Ohio
Feiman, Edward M., 212 First Nat'l Bk. Bldg., Canton 2, Ohio
Hargett, Edgar R., 4 West Main St., Springfield, Ohio
Levey, Mark R., 830 Tegler Bldg., Edmonton, Alberta, Can.
Maumenee, Alfred E., Johns Hopkins Hosp., Baltimore 5, Md.
Munoz-MacCormick, Carlos E., Monterrey Bldg., Park Terrace, Santurce, Puerto Rico
Ogus, Alfred, Norton General Hospital, Norton, Va.
Perry, James S., McCartney Drug Store, Bryan, Texas
Podboy, August J., 211 S. George Street, York, Pa.
Reeh, Merrill J., Station Hospital, Randolph Field, Texas
de Rezende, Nilson T., Esteves Jr. 68, Rio, Brazil, S. A.
de Roetth, Andrew F., 508 Old Nat'l Bk. Bldg., Spokane 8, Wash.
Rowland, Alan L., 1031 Bank of America Bldg., San Diego, Calif.
Seelye, Norman Lee, 36 North Ayer St., Harvard, Ill.
Winston, Samuel H., Reeves Bk. Bldg., Dover, Ohio
Zarbaugh, Guy F., I.O.O.F. Bldg., Deadwood, South Dakota

Necrology

IN MEMORIAM

John Finch Barnhill, Miami Beach, Florida

Joseph C. Beck, Chicago, Illinois

Theodore S. Blakesley, Kansas City, Missouri

Alice Gertrude Bryant, Boston, Massachusetts

Robert Emmett Buckley, New York, N. Y.

Donald Malcolm Campbell, Detroit, Michigan

Harry B. Davis, Kansas City, Mo.

Ewing Wilbur Day, Pittsburgh, Pa.

Dred Rumley Dorente, Ada, Oklahoma

Joseph F. Duane, Peoria, Illinois

Louis Semon Dunn, Philadelphia, Pa.

Curtis Clyde Eves, Philadelphia, Pa.

John Daniel Fouts, Dayton, Ohio

William Fowler, Detroit, Michigan

Arthur Henry Geiger, Chicago, Illinois

Henry William George, Middletown, Pa.

Allen Greenwood, Boston, Massachusetts

James Beaty Griffith, Washington, D. C.

Frank Allen Hartley, Springfield, Ohio

Sergius M. Ingerman, New York, N. Y.

Edward Jackson, Denver, Colorado

Wilson Johnston, Portland, Oregon

Robert McKee Lapsley, Keokuk, Iowa

Jonathon Brown Loring, Chicago, Illinois

Carl Charles McClelland, Detroit, Michigan

S. Hanford McKee, Montreal, Canada

Richmond McKinney, Memphis, Tennessee

Willard Geist Mengel, Camden, New Jersey

Stanley George Miner, Detroit, Michigan

Stewart Brown Muncaster, Washington, D. C.

Edward Powers Norcross, Highland Park, Illinois

Eugene P. Norwood, Corsicana, Texas

Samuel Poindexter Oast, New York, N. Y.

Luther Crouse Peter, Philadelphia, Pa.

William Aubrey Poole, Lexington, Kentucky

Howard Thomas Robinson, Cumberland, Maryland

Joseph M. Rosenthal, Philadelphia, Pa.

George Samuel Row, Indianapolis, Indiana

Abraham Noah Schiller, New York, N. Y.

Louis Joseph Sebert, Toronto, Canada

Walter J. Sener, Media, Pennsylvania

Frederick Casimir Simon, St. Louis, Missouri

Harry Bayliss Stokes, Omaha, Nebraska

James Craig Strong, Leadville, Colorado

Edward Franklin Stroud, Corpus Christi, Texas

Frank Vinsonhaler, Little Rock, Arkansas

Carl B. Wagner, Chicago, Illinois

C. B. Walker, Los Angeles, California

Nelson Shelly Weinberger, Sayre, Pa.

Walter Joseph Wherry, Omaha, Nebraska

JOHN FINCH BARNHILL

Doctor Barnhill was born on January 2, 1865, on a farm near Nora, Indiana. He received his early grammar school and high school education in Westfield, Indiana. He was married to Celeste Terrell who survives him. They had no children.

Doctor Barnhill began his career as a school teacher. He later entered the Central College of Physicians and Surgeons, which eventually became one of the component parts of Indiana University School of Medicine as it is today. He was graduated in 1888.

After practicing general medicine a short time in Indianapolis he became interested in otolaryngology. He did not, however, simply announce that this was the special field of work he had chosen and begin practicing it without any preparation, as many men did at that time. He sought medical centers where he could receive this training. In our own country he found this instruction in Chicago, New York City and Philadelphia; and abroad he found it in London, Paris, Berlin and Vienna.

Doctor Barnhill was president of his local medical society in 1924, of the American Laryngological, Rhinological and Otological Society in 1928, the American Academy of Ophthalmology and Otolaryngology in 1931, the American Laryngological Association in 1937, and was Chairman of the Section of Rhinology, Otology and Laryngology of the American Medical Association in 1903-1904. He was a Fellow of the American College of Surgeons.

Doctor Barnhill took a very keen interest in general medical affairs. He was always interested in the welfare of the younger men who were working in his chosen specialty, and he never failed to promote their welfare if they were deserving of such promotion.

His interest in medical education dated from the time he became an assistant in the Department of Otolaryngology in Indiana University School of Medicine and continued to his demise. Doctor Barnhill was chairman of this department; he was chairman of the Department of Head and Neck Surgery, and he developed an anatomical and clinical course in otorhinolaryngology. Out of this course came his work on the anatomy of the head and neck which was unique in that he presented it in monologue style. One would have to be a student and co-worker of his to appreciate this work fully.

Doctor Barnhill, with Doctor Ernest deW. Wales, published a work on otolaryngology. He read numerous papers before the special societies, many of which were later published. He never feared to express his own opinions in a discussion. In the literary fields his hobby was along historical lines, and one of his outstanding productions was "Hatching the American Eagle." He was granted the degree of Doctor of Laws by his alma mater, Indiana University, in 1929.

He was one of the founders of the present Indiana University School of Medicine. He was responsible for obtaining the endowment for the Robert Long Hospital which was the first teaching hospital of the medical school center.

All the members of his staff in the University liked to address him as "Uncle Jeff" and he was quite fond of this bit of familiarity when so addressed. He enjoyed a good story and liked to tell one. During his last years he was thrilled when he could be present at the "Barnhill" dinners given for him at the Academy meetings by the fellows who had taken his anatomical and clinical courses.

Doctor Barnhill was physically handicapped throughout his life by being afflicted with asthma. During the last ten years polyarthritis was added, from which he suffered intense pain. Despite these afflictions he lived to the ripe age of 78 years. He died on March 10, 1943, at Miami Beach, Florida, where he had made his home for the past ten years.

JOSEPH C. BECK

With the death of Joseph C. Beck, on October 20, 1942, American otolaryngology lost one of its pioneers as well as one of its strongest characters.

He was born in Bohemia, September 26, 1870, and the story of his life rivals that of fiction. His career personifies the opportunities of America that are open to those who do truly and earnestly seek them. His childhood was not carefree — his mother died when he was but two years old and his father when he was nine. Perhaps it was the memories of his youth that made him kind and sympathetic and so very human. The story of his childhood, of his youth in America, of his determination to study medicine and of his medical career are well told in his book "Fifty Years in Medicine."

After graduating in 1895 from the University of Illinois, he became associated with his brother, Carl, in the practice of medicine. Three years later he decided to specialize, and it is interesting to note that in doing so he decided first to learn about pathology. He went to Prague where he devoted a year to the study of pathology and two years more in preparing for the specialty of otolaryn-

gology. After returning to Chicago his career was one of steady progress. He became associated with his brothers, Emil and Carl, following which the North Chicago Hospital became the Mecca of all visiting otolaryngologists. His interest in pathology continued, and, as a charter member of the American Board of Otolaryngology, he was instrumental in making graduate training in otolaryngology "pathology conscious."

He was a pioneer, too, in former borderline specialties such as plastic surgery, endoscopy and treatment of tumors of the head and neck.

He was a teacher. During his entire medical career he taught. He was associate professor in otolaryngology from 1901-1932 at the University of Illinois College of Medicine; professor of otolaryngology from 1902-1914 at the Chicago Eye, Ear, Nose and Throat Hospital, and he gave many instructional courses at annual sessions of the American Academy of Ophthalmology and Otolaryngology. At every opportunity he gave to his students freely of his experiences.

He encouraged reading of the literature. His reading clubs (there were five) met once a month, and Doctor Beck never missed a meeting. There the literature of all languages was abstracted and reviewed.

During World War I Doctor Beck was in charge of the hospital sponsored by the American Red Cross for the Czechoslovakian Government. He served until after the Armistice. He was awarded the Serbian Cross and the Czechoslovakian Revolutionary Medal. Doctor Beck was discharged with the commission of Lieutenant-Colonel in the United States Medical Reserve Corps. After the War his interest in military affairs continued. He became a member and later a Commander of the Medical Post of the American Legion in Chicago.

Doctor Beck was the author of eighty or more medical papers, and he perfected many scientific instruments.

He was a member of all, and a past president of most, of the American otolaryngological societies. In 1931 he was chosen Honor Guest of the American Academy of Ophthalmology and Otolaryngology, and he was made a member of the Honor Society of the Academy.

Doctor Beck (to most of his friends and students, "Doctor Joe") loved life and youth. He enjoyed a good story. The movies, music, art, sports and at one time "His Farm" were his hobbies. His influence was widespread. His intense interest, enthusiasm and kindness were genuine. If you were a friend of his you knew it; if there was any doubt about it, you likewise knew it. He was fearless and intellectually honest.

As an expert diagnostician, for ten months he calmly watched his own decline toward death. His passing leaves a great void in our ranks. "Doctor Joe" lived his own obituary:

"To those who knew thee not, no words can paint;
And those who knew thee, know all words are faint."

THEODORE S. BLAKESLEY

Doctor Blakesley was born in 1878. He received his medical degree from Rush Medical College in 1902 following which he engaged in postgraduate study in ophthalmology and otolaryngology in medical schools in France, Austria and Germany. He practiced his specialties in Kansas City, Missouri.

Doctor Blakesley was a member of the staff of Kansas City General Hospital and St. Joseph Hospital, and was assistant clinical professor in ophthalmology at the University of Kansas.

In 1925 he was certificated by the American Board of Otolaryngology. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association and the American College of Surgeons as well as state and local medical societies.

During World War I he was the second flight surgeon to be assigned to foreign duty, and he attained the rank of Major while in the Medical Corps of the U. S. Army.

Doctor Blakesley was widely known as an amateur magician, and he became so proficient in the art of legerdemain that Houdini, when he came to Kansas City, would visit him at his home.

His death occurred on July 14, 1943, in Ohio State Hospital, Columbus. He is survived by his wife, a daughter, a brother and a sister.

ALICE GERTRUDE BRYANT

Doctor Bryant was born in Boston on April 27, 1862, the daughter of Charles B. and Elizabeth S. Bryant.

She was a student at Vassar College, Massachusetts Institute of Technology and Women's Medical College of Pennsylvania before receiving her degree in medicine from the Women's Medical College of New York Infirmary in 1890. She interned at the New England Hospital for Women and Children, Boston.

Doctor Bryant was otologist and laryngologist to the Trinity Dispensary from 1893-1898, to the Vincent Memorial Hospital from 1893-1898, to the Pope Dispensary from 1898-1909 and to the New England Hospital for Women and Children from 1898-1909. Later she held similar positions on the courtesy staff of the New England Deaconess Hospital, Boston.

She was a Life member of the American Academy of Ophthalmology and Otolaryngology, a Fellow of the American College of Surgeons, member of the American Medical Association, the British Association for the Advancement of Science, the American Association for the Advancement of Science, Fellow of the Institute of Hygiene of Great Britain, member of Gorgas Memorial Institute of Tropical and Preventive Medicine, New England Rhinological and Otolaryngological Society and Massachusetts Medical Society.

During World War I Doctor Bryant was a member of the Volunteer Medical Service Corps No. 2852.

She was the author of many medical and scientific articles. She discontinued performing surgery upon her retirement from the hospital. Despite her 80 years, however, she was in active office practice until the last few months of her life.

Doctor Bryant was of a shy, retiring nature and one of the pioneer women in her specialty. She was a great student, not confining her studies to medicine, but taking a keen interest in art, literature, politics and everything which concerned the higher education and betterment of women. She was an ardent student, never so happy as when in her study surrounded by her books — reading, studying and writing often into the late hours of the night.

Her death occurred in the Palmer Memorial of the New England Deaconess Hospital, Boston, on July 25, 1942.

ROBERT EMMETT BUCKLEY

Doctor Buckley was born in New Britain, Connecticut, on December 14, 1888.

He was graduated from Yale University Medical School in 1911 following which he served a one year's internship in St. Mary's Hospital, Brooklyn, and began the practice of medicine in New York City.

Doctor Buckley was director of surgery in laryngology at the Manhattan Eye, Ear and Throat Hospital, consulting otolaryngologist to the Misericordia Hospital and Roosevelt Hospital, New York, to St. Luke's Hospital, Newburgh, New York, and the Yonkers (N. Y.) Professional Hospital. At one time he was assistant professor of laryngology and professor of clinical otology at the New York Post-Graduate Medical School, Columbia University.

He was certificated by the American Board of Otolaryngology in 1925 and was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the American Laryngological Association, the American Laryngological, Rhinological and Otological Society and the Medical Society of the State of New York.

He served with the Roosevelt Hospital Unit in France during World War I.

Doctor Buckley's death occurred on July 25, 1942. He is survived by his wife and two daughters.

DONALD MALCOLM CAMPBELL

Doctor Campbell was born in Wardsville, Ontario, Canada, on December 12, 1863.

He attended public schools and collegiate institutes in Windsor, Ontario, and received his medical degree in 1885 from the Detroit College of Surgeons (Wayne University). He engaged in postgraduate study in ophthalmology and otolaryngology in Edinburgh, London, Dublin, New York and Chicago. He was made a licentiate of the Royal College of Surgeons and of the Royal College of Physicians (Edinburgh) and of the Royal College of Surgeons and the Royal College of Physicians (Glasgow). He was certificated by the American Board of Ophthalmology in 1921 and by the American Board of Otolaryngology in 1925.

Doctor Campbell was consulting ophthalmologist at the City of Detroit Receiving Hospital and Harper Hospital, Detroit, since 1892. He was clinical assistant to the chair of diseases of the eye and ear at Wayne University from 1888-1893, lecturer on diseases of the eye and ear from 1893-1894 and professor of diseases of the eye and ear from 1907-1908. He was professor of ophthalmology and otolaryngology at Wayne University from 1913-1914 and professor of ophthalmology at the University from 1913-1939. In 1939 he was made professor emeritus.

Doctor Campbell was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the American Laryngological, Rhinological and Otological Society and the American Otological Society.

His interests other than medical were in travel and sports.

Doctor Campbell died in Detroit on August 25, 1942. He is survived by two daughters and one son, Donald M. Campbell, Jr., of Detroit.

EWING WILBUR DAY

Doctor Day was born in Deerfield, Ohio, on November 1, 1862, the son of Edgar M. and Frances Reed Day. A graduate of Allegheny College (B. A. 1884, M. A. 1887) and Georgetown University (M.D., 1889), he obtained his postgraduate training in otolaryngology in Vienna.

Doctor Day practiced otolaryngology in Pittsburgh for 40 years, serving on the staffs of a number of Pittsburgh hospitals. He was a founder of the Pittsburgh Eye and Ear Hospital and its chief of staff from 1908 until his retirement in 1928. For many years he was the professor of otolaryngology at the University of Pittsburgh and since 1928 had been professor emeritus.

He was a Life member of the American Academy of Ophthalmology and Otolaryngology, founder and past president of the Pittsburgh Academy of Medicine, past president of the Allegheny County Medical Society, past president of the American Otological Society, past president and treasurer of the American Laryngological, Rhinological and Otological Society and a member of International Collegium Oto-Rhino-Laryngolicum. In World War I he served as a Major and Lieutenant Colonel with the United States Army.

Doctor Day was a founder and a charter member of the Triological Society at its inception in 1895. He was president in 1908 and a member of the Council for 40 years. It is as the treasurer of the society, 1901-1907 and 1909-1937, however, that Doctor Day will be long remembered. Through his single-handed efforts and shrewd judgment, he made the financial status of the society an enviable one. No member has ever had the interest of the society more at heart and none has contributed more to its stability and welfare.

His activities included membership in the Sons of the American Revolution, the Order of Patriots and Founders, the American Legion, the Military Circle of the World War, the Duquesne Club and the University Club of Pittsburgh. In religion his affiliation was with the Episcopal Church; in politics, he was a Republican.

Since his retirement, Doctor Day made his home in Provincetown, Massachusetts.

His death occurred on November 24, 1942. Surviving him are his wife, Annie Mosier Day and three sons, Major Ewing W. Day, II, Dr. Kenneth M. Day and Percival E. Day.

DRED RUMLEY DORENTE

Doctor Dorente was born on October 11, 1880, in Parker, Pennsylvania.

He attended public schools in Allegheny, Pennsylvania, and graduated from the College of Physicians and Surgeons, Dallas, Texas, in 1907. He received his medical degree from the Chicago College of Medicine and Surgery (Loyola University) in 1913, following which he engaged in postgraduate study in otolaryngology and ophthalmology at the New York Post-Graduate School and Hospital.

He practiced his specialties in Fort Smith, Arkansas, where he served as a member of the staff of Sparks' Memorial Hospital and as vice president of the executive staff of St. Edwards Mercy Hospital. He was chief oculist for the Fort Smith Western Railroad and division oculist for the Frisco Railroad and the Missouri Pactific Railroad.

Doctor Dorente was certificated by the American Board of Otolaryngology in 1917. He was a Senior member of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association, Fellow of the American College of Surgeons and a member of state and local medical societies.

He was made a Captain in the United States Army during World War I. Doctor Dorente was a 32nd Degree Mason.

He died in Ada, Oklahoma, on June 17, 1942, and is survived by two sisters and one brother.

JOSEPH F. DUANE

Doctor Duane was born on October 12, 1880, in Auburn, New York.

He received his preliminary education in Peoria, Illinois, and his medical degree from Rush Medical College in 1903. The University of Notre Dame honored him with the degree of Litt. B.

In 1906, Doctor Duane began the practice of internal medicine in Peoria. He later engaged in postgraduate study in ophthalmology in Vienna, Berlin, London, New York and Boston, and in 1915 began to practice his specialty in Peoria. He was a member of the staff of St. Francis Hospital.

Doctor Duane was a Senior member of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association, the Illinois State Medical Society, the Peoria County Medical Society and the Chicago Ophthalmological Society.

His church affiliation was Roman Catholic. His chief hobby was his profession. He was an ardent golfer and traveled extensively.

Doctor Duane's death occurred on January 21, 1943, in Peoria. He is survived by his wife, a daughter, Mrs. Valerie D. Flegel and a son, Thomas D. Duane, who is a medical student at Northwestern University.

LOUIS SEMON DUNN

Doctor Dunn was born on August 27, 1886, in Rochester, New York.

He attended the public schools in Rochester and the University of Chicago. He received his medical degree from Medico-Chirurgical College, Philadelphia, in 1907.

Doctor Dunn began the practice of general medicine and surgery in Chester, Pennsylvania, in 1908. In 1916, he engaged in special training in otolaryngology at Johns Hopkins University and medical institutions in Berlin, Vienna and Heidelberg, following which he returned to Chester, Pennsylvania. In 1920, he moved to Philadelphia where he practiced his specialty until his death.

He was associate professor of laryngology and otology at the Graduate School of Medicine, University of Pennsylvania. He gave many lectures on pathology of the sinuses. Doctor Dunn was associated with the late Dr. Ross Hall Skillern and then with Dr. George M. Coates, of Philadelphia. He served on the staffs of St. Agnes, Jewish and Mt. Sinai hospitals in Philadelphia.

He was certificated by the American Board of Otolaryngology in 1925. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, Fellow of the American College of Surgeons, member of the Pennsylvania State Medical Society, the Philadelphia County Medical Society and the Philadelphia Laryngological Society. He was a life member of the Ancient Accepted Scottish Rites, life member and alumni adviser to the Phi Alpha fraternity, member of the American Legion and of the publication committee of the "Hebrew Physician."

In World War I Doctor Dunn served as a surgeon from June 1918 to June 1919 with the commission of First Lieutenant, Base Hospital No. 89, France. In 1930 he was commissioned Major in the Medical Reserve Corps of the United States Army.

A scholar, a linguist, an authority on Hebrew lore and a public speaker of note, Doctor Dunn delivered many historical lectures, addressed religious groups of all creeds and gave unstintingly of his time to civic and welfare work. Of him Dr. John Deaver, surgeon, said, "He represents the highest standards of the profession."

Doctor Dunn was the author of many medical papers concerning pathology of the sinuses.

His death occurred in Philadelphia on June 16, 1942. His wife, Sari R. Dunn, and daughter, Felice Constance, survive him.

CURTIS CLYDE EVES

Doctor Eves was born in Millville, Pennsylvania, in 1874. His father was Joseph C. Eves, postmaster at Millville, and his mother, Charlotte Heacock Eves.

Doctor Eves was a Birthright member of the Society of Friends and a lifelong member of the Millville, Pennsylvania, Meeting. This religious upbringing and continued connection greatly influenced his whole life.

His early education was obtained at the George School, Bucks County, a Hicksite Friends Boarding School. His interest in this school continued throughout his life, and for many years he served as one of its trustees. In fact, his position as physical director of the George School enabled him to obtain his medical education. He spent two years at the College of Vermont before coming to Philadelphia to enter the Medico-Chirurgical College where he was graduated in 1905. He interned at the Episcopal Hospital. After his internship he immediately took up the study of otolaryngology, going to the New York Eye and Ear Infirmary where he was resident for two years. He began to practice his specialty in Philadelphia in 1910. Later, in 1926, he spent some time studying in Vienna.

Doctor Eves was chief of otolaryngology at the Episcopal Hospital, resigning in 1915. Through his association with Dr. Francis R. Packard he became assistant in the otolaryngological department of the Pennsylvania and Bryn Mawr hospitals, and on the retirement of Doctor Packard was elected chief of these services in both hospitals in 1931. He retired from active hospital work in 1937, being appointed consulting otolaryngologist to the Pennsylvania and the Bryn Mawr hospitals at that time.

Doctor Eves was certificated by the American Board of Otolaryngology in 1925. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, American Laryngological Association, American Otological Association, American Broncho-Esophagological Society and American Laryngological, Rhinological and Otological Society. He was

elected a Fellow of the American College of Physicians in 1912. He was chairman of the Section on Otolaryngology of the American Medical Association in 1937-1938 and served on its executive committee until the time of his death.

In his vacational periods he turned to the out-of-doors. He was an enthusiastic fisherman, and large parts of his vacations were spent on the salmon streams of Canada. For the past few years he was a member of the Swiftwater Preserve. He also enjoyed and played a good game of golf and was a member of the Doctors' Golf Association. He belonged to the Union League and the Philadelphia Country Club.

Although Doctor Eves was not a prolific writer, his published articles were real contributions to the specialty. Before the days of Chevalier Jackson, in Philadelphia, he was one of the few Philadelphians who really practiced bronchoscopy. He was a skillful operator and a careful and accomplished clinician, thoroughly cognizant of the progressive improvements in diagnosis and treatment.

Doctor Eves was admired for his professional ability and for his inherent and intrepid sense of right.

His death occurred in Philadelphia on September 22, 1942. His wife, Glodean S. Eves, survives him.

WILLIAM FOWLER

Doctor Fowler was born in Zorra, Ontario, on January 10, 1886. He received his medical degree from Michigan College of Medicine and Surgery in 1903, following which he engaged in postgraduate study in ophthalmology and otolaryngology at the Royal Infirmary of Edinburgh. Doctor Fowler practiced his specialties in Alderson, Oklahoma, before moving to Detroit.

He was certificated by the American Board of Otolaryngology in 1925. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association and a Fellow of the American College of Surgeons. He was a member of the staff of Grace Hospital, Detroit.

Besides his affiliation with medical societies, Doctor Fowler was a member of St. Andrews Scottish Society, the Maccabees, Odd Fellows and also the Michigan Poetry Society. He was especially interested in poetry, music and Scottish folk lore. He was the author and composer of an operetta entitled "Bonnie Prince Charlie" and a musical selection "Lovely Scotland." His "Poems by William" were published in the Journal of the American Medical Association.

Doctor Fowler died on July 13, 1942, in Ferndale, Michigan. Surviving are his wife, Evelyn B. Fowler, and a daughter, Dorothy Jean.

ARTHUR HENRY GEIGER

Doctor Geiger was born on January 6, 1878, in Chicago.

He received his medical degree from the Medical School of the University of Illinois in 1900 following which he served as assistant to Dr. Christian Fenger of Chicago. He engaged in postgraduate study in otolaryngology in Berlin, Vienna and London in 1908, 1911 and 1912.

Doctor Geiger was clinical professor in otolaryngology at the Chicago Polyclinic Postgraduate School from 1912 to 1922 when he resigned to become Chairman of the Illinois Board of Medical Examiners, which position he held until the time of his death. He served on the staffs of the Polyclinic, Henrotin, Alexian Brothers, Evangelical Deaconess and Illinois Masonic hospitals, being elected president of the latter in 1943.

He was certificated by the American Board of Otolaryngology in 1925. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association and state and local medical societies. For many years Doctor Geiger served on the Council of the Chicago Medical Society and as a member of the House of Delegates of the Illinois State Medical Society. On several occasions he was a member of the House of Delegates of the American Medical Association.

Doctor Geiger was especially interested in the affairs of the Medico-Legal Committee of the Illinois State Medical Society of which he was a member. He was a keen observer, with remarkable analytical powers to discern and solve complicated problems. These qualifications made him invaluable as a member of the Medico-Legal Committee where sound judgment is required.

At the beginning of World War I he was commissioned a First Lieutenant in the Medical Corps of the U. S. Army. At the time of his discharge from the Army he had attained the rank of Lt. Colonel in the Medical Reserve Corps.

Doctor Geiger sketched, painted and was a member of the Physicians' Society (Art). His religion was Episcopalian. He was a staunch Republican. He was a member of the Medical Post of the American Legion.

His death occurred on May 12, 1943. He is survived by his wife, Mrs. Margaret Geiger.

HENRY WILLIAM GEORGE

Doctor George was born in Mountain Top, Pennsylvania, on May 27, 1878. He attended Muhlenberg College and received his medical degree from Jefferson Medical College in 1900. Doctor George interned at the Samaritan Hospital, Philadelphia, from 1900-1901, following which he practiced general medicine in Middletown, Pennsylvania.

From 1916-1920 he was a resident at Wills Hospital, Philadelphia, and he engaged in graduate study in ophthalmology and otolaryngology at Harvard Medical School, the University of Pennsylvania Graduate School and the University of Vienna Graduate School.

He was certificated by the American Board of Ophthalmology in 1937. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, Ophthalmological Society of the United Kingdom, Harrisburg Academy of Medicine, Pennsylvania Railroad Surgeons Association, Central Pennsylvania E. E. N. and T. Association and the Dauphin County Medical Society.

He was ophthalmologist on the staff of the Keystone Hospital, Harrisburg, and ophthalmologist to the Emans Orphans Home and Odd Fellows Home there.

He was Ruling Elder in the Middletown Presbyterian Church and had been superintendent of the Sunday school for 30 years. He made numerous trips to Europe and the Near East and was the author of an essay entitled "Ophthalmology in the Near East."

Doctor George died on July 7, 1942, in Philadelphia. Surviving him are three daughters and a son, Dr. F. P. George, of Harrisburg, Pennsylvania.

ALLEN GREENWOOD

Doctor Allen Greenwood was born in Chelsea, Massachusetts, March 1, 1866. He received his M.D. degree from Harvard Medical School in 1889; was house officer in the Boston City Hospital 1888-1890; instructor in Waltham Training School 1890-1901; bacteriologist Waltham Board of Health 1894-1898; instructor in ophthalmology Harvard Medical School 1904-1905; ophthalmic surgeon to the Boston City Hospital 1901-1918; since, consulting ophthalmologist; visiting surgeon 1895-1900, visiting ophthalmic and aural surgeon since 1900, Waltham Hospital; consulting ophthalmic surgeon Massachusetts Eye and Ear Infirmary and Beth Israel Hospital (Boston), Union Hospital (Framingham), Milford Hospital (Mass.); lecturer on ophthalmology Harvard Graduate School of Medicine; professor of ophthalmology Tufts Medical School.

Doctor Greenwood was appointed acting assistant surgeon of the U. S. Army August 22, 1898, and was assigned to the 19th U. S. Infantry, Ponce, Puerto Rico. He was honorably discharged October 19, 1898. During the World War he was Honorary Lieutenant Colonel in the Royal Army Medical Corps with the British Expeditionary Force in France in 1916; in 1917 he became a member of the sub-committee on ophthalmology of the General Medical Board of National Defense and was on duty in the Surgeon General's office as Command Major in the Medical Reserve Corps. He was made Lieutenant Colonel in the Medical Corps and consultant in ophthalmology for the American Expeditionary Force in 1918; Colonel of the Medical Reserve Corps, U. S. Army, in 1919. Following the War he compiled from the records of the Surgeon General's office the report on ophthalmology (American Expeditionary Forces). The Medical Department of the U. S. Army in the World War, Vol. XI, Part 2, Section IV, pp. 659-728.

He was chairman of the Section on Ophthalmology of the American Medical Association in 1919-1920; president of the American Academy of Ophthalmology and Otolaryngology in 1918; chairman of the Honor Society of the Academy in 1941; president of the New England Ophthalmological Society in 1903 and of the American Ophthalmological Society in 1942. He was a member of the Association for Research in Ophthalmology, Inc., and a fellow of the American College of Surgeons. He was a member of the Board for Ophthalmic Examinations (now the American Board of Ophthalmology) since 1921.

Doctor Greenwood died on October 18, 1942, in Miami, Florida.

JAMES BEATY GRIFFITH

Doctor Griffith was born in Severn, Maryland, on July 17, 1890.

He attended high school in Rockville, Maryland, obtained his B.A. degree from Southern College, Florida, his M.A. degree from the University of Florida and his medical degree from the University of Tennessee in 1918. From 1918-1919 Doctor Griffith interned at Memphis General Hospital. He was a resident at the New York Eye and Ear Infirmary in 1919. His specialty was ophthalmology.

Doctor Griffith was certificated by the American Board of Ophthalmology in 1936. He was a member of the American Academy of Ophthalmology and Otolaryngology, the

American Medical Association, the Washington Ophthalmological Society and the Washington Medical and Surgical Society.

He was chief surgeon at the Episcopal Eye and Ear Hospital, Washington, D. C., a member of the staff of the Emergency and Georgetown hospitals, Washington, D. C. and assistant professor of ophthalmology at Georgetown University Medical School.

Doctor Griffith's hobbies were fishing, hunting, skeet shooting and farming.

His death occurred in Washington, D. C. on May 6, 1942. His wife, mother and two sisters survive.

FRANK ALLEN HARTLEY

Doctor Hartley was born in Union County, Ohio, on November 27, 1870.

He was graduated from the Starling Medical College in 1898 and for a few years thereafter practiced in Byhalia and in Rosewood, Ohio. He engaged in postgraduate study in ophthalmology and otolaryngology at the Polyclinic Hospital in Philadelphia in 1907-1908, and at the University of Vienna in 1909-1910. Doctor Hartley came to Springfield, Ohio, in 1910 and limited his practice to ophthalmology and otolaryngology there for the next 33 years.

He was an active and industrious member of the staff of the City Hospital until 1939 when he became an emeritus member. He was attending ophthalmologist and otolaryngologist on the staff of the Rickley Memorial Hospital during his later years.

Doctor Hartley was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the Ohio State Medical Society and the Clark County Medical Society.

His death occurred on January 18, 1943, in Springfield. He is survived by his wife, Della M. Hartley.

SERGIUS M. INGERMAN

Doctor Ingerman was born in Russia on August 15, 1868.

He received his early education in Odessa. As a youth he joined the famous Narodnaya Volia, precursor of the revolutionary organizations which ultimately brought about the downfall of the Czarist government. Because of his activities against the old regime, he was compelled to leave Russia, going to Switzerland where he entered the medical school at the University of Bern, from which he was graduated in 1889. Together with his wife, Dr. Anna Ingerman, he emigrated to America in 1891, making his home in New York City. He became a practicing physician there, specializing in eye, ear, nose and throat.

At one time Doctor Ingerman was an instructor in ophthalmology at the New York University Medical College. He was consulting ophthalmologist at Sydenham and Beth Israel Hospitals in New York City. For many years he was otorhinolaryngologist to the Tuberculosis Sanitarium of Workmen's Circle at Liberty, New York.

He was a Life member of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association, the International Medical Club and the Medical Society of the State of New York. He was a past president of the Russian Medical Society.

His death occurred on February 18, 1943, in New York City. Surviving him are his daughter, Dr. Eugenia Ingerman, a son-in-law, Bela Low, a metallurgical expert for the War Production Board, and a grandson, Francis Low, an instructor at Princeton University, now in the United States Air Force.

EDWARD JACKSON

Because of his conspicuous services, his advanced age and his remarkably prolonged activity as thinker and teacher, Doctor Jackson had for years been regarded as the dean of ophthalmology. He was with us, genial and lively as ever, at the Chicago meeting October last. Two weeks after that meeting he was on his death-bed. No man has been more beloved by his colleagues, and there has been none whose passing was more widely and sincerely regretted.

Doctor Jackson was born in West Goshen, Pennsylvania, March 30, 1856. Before studying medicine he had obtained the degree of Bachelor of Science in civil engineering from Union College, New York. After receiving the medical degree from the University of Pennsylvania, in 1878, he practiced general medicine for a short time at West Chester, Pennsylvania. That same year he married Jennie L. Price. He moved to Denver in 1894 on account of his wife's health, returned to Philadelphia after her death in 1896, and in 1898 again moved to Denver, where he married Emily Churchman.

In 1905 he became professor of ophthalmology in the Medical Department of Colorado University.

In 1890 he published "Essentials of Diseases of the Eye"; in 1895, his "Skiascopy and Its Practical Application" (a profound scientific study of this objective test); and in 1900 "A Manual of the Diagnosis and Treatment of Diseases of the Eye." In addition to many

hundreds of miscellaneous medical essays and editorials, he wrote the section on eye muscle operations in Wood's System of Ophthalmic Operations; that part of the chapter on ocular muscles in Wood's American Encyclopedia of Ophthalmology, which deals with operations on the extrinsic muscles, and the chapter on skiascopy in the American Encyclopedia of Ophthalmology.

Doctor Jackson was president of the American Academy of Ophthalmology and Otolaryngology in 1904, chairman of the Section on Ophthalmology of the American Medical Association in 1894, and president of the American Ophthalmological Society in 1912.

At first, with financial aid from Doctors de Schweinitz and Schneideman, but chiefly on his own financial responsibility and with the burden of an annual deficit, he founded the Ophthalmic Year Book, and edited it from 1904 to 1923, inclusive. He was instrumental in creating the American Journal of Ophthalmology, third series, which took the place of a number of separate periodical publications on ophthalmology; and he edited that Journal from 1918 to 1927, inclusive. He continued to act as consulting editor of the Journal until his death, writing one or more editorials almost every month.

His most important activities were related to improvement in education for ophthalmic practice, with a plan for certification of American ophthalmologists. These efforts culminated in the creation, in 1915, of the American Board for Ophthalmic Examinations (now the American Board of Ophthalmology), which was the first organization of its kind in the United States. He was the first president of the Board and served for eleven years as one of its members. He had already persuaded the University of Colorado to follow the example of Oxford University, England, in the creation of an advanced medical degree in ophthalmology. He utilized the energy of his colleagues in the Colorado Ophthalmological Society (of which he had been one of the founders in 1899) and in the Colorado Otolaryngological Society in conducting for a number of years the Colorado Summer Course in Ophthalmology and Otolaryngology. This enterprise pointed the way for a number of similar courses in various parts of the United States, especially the mid-winter course in Los Angeles.

Doctor Jackson was for several years on the Board of Directors of the National Society for the Prevention of Blindness, whose Leslie Dana medal he received in 1925, and he was for some years chairman of the Colorado State Commission for the Blind.

An incessant worker, Doctor Jackson placed his duty as a thinker and teacher ahead of all else. He was particularly interested in aiding the development of the younger ophthalmologists. He lived simply, even austerely, always loyal to the principles of the Society of Friends, into which he had been born.

ROBERT McKEE LAPSLEY

Doctor Lapsley was born in Clark County, Missouri, in 1870.

He attended the College of Physicians and Surgeons, Keokuk, Iowa, and completed his medical studies at Rush Medical College from which he obtained his M. D. degree in 1891. His preliminary postgraduate study in ophthalmology and otolaryngology consisted of a clinical course at Rush Medical College and special training at the Manhattan Eye and Ear Infirmary, where he served as clinical assistant. In 1898 Doctor Lapsley went abroad to study at the Royal London Ophthalmic Hospital and the Central London Throat and Ear Hospital where he served as clinical assistant in addition to attending clinics. Later he engaged in postgraduate study in Vienna.

He was professor of ophthalmology and otology at Keokuk Medical College. He practiced continuously in Keokuk since 1894 and served as president of the staff of St. Joseph Hospital since 1928.

Doctor Lapsley was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, Fellow and charter member of the American College of Surgeons and a member of state and local medical societies.

Aside from his prominence in the medical profession, Doctor Lapsley was also active in religious, civic, fraternal and social circles.

His death occurred on January 5, 1943. His wife survives him.

JONATHAN BROWN LORING

Doctor Loring was born in Sherbrooke, Quebec, Canada, on January 12, 1859.

He studied at McGill University in Montreal and graduated in 1881 with the degrees M.D. and C.M. While there, he was under the direct teaching and influence of William Osler. He spent two years in postgraduate study in ophthalmology at the Royal College of Surgeons, London, England, and obtained the degree M. R. C. S., England, in 1883. After this course of study he came to Chicago to practice his specialty.

He was assistant eye surgeon on the staff of the Illinois Charitable Eye and Ear Infirmary, 1894-1909; assistant professor, clinical

ophthalmology, University of Illinois, 1909-1913; clinical professor of ophthalmology at the same institution, 1913-1918.

Doctor Loring was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, Institute of Medicine of Chicago, Chicago Ophthalmological Society, of which he was president in 1924, Physicians Club of Chicago, Society of Medical History of Chicago and a Fellow of the American College of Surgeons. He was also honorary member of Alpha Kappa Kappa and Alpha Omega Alpha fraternities.

He was always a good student and a keen and careful diagnostician. Although a good surgeon, he was more an ophthalmic physician, and all surgical cases were studied in minute detail before any surgical operation was performed. As a teacher, he was an advocate of small group clinical instruction and in that he was a master in imparting knowledge.

He continued in practice until February, 1942, when he retired on account of ill health. His death occurred in Chicago, November 6, 1942. His wife, Mary Armitage Loring, and a daughter survive him.

CARL CHARLES McCLELLAND

Doctor McClelland was born on October 17, 1879, in Jefferson, Ohio.

He attended Jefferson high school and Michigan State Teachers College, and he received his degree in medicine from the University of Michigan.

He began his career as an educator, serving successively as principal of the Eaton Rapids high school, Benton Harbor high school and superintendent of the Benton Harbor schools. Turning to the study of medicine in 1906, he entered the University of Michigan where he obtained his degree in 1910. He studied abroad at the London Eye and Ear Hospital and in Vienna before coming to Detroit in 1912 when he began his practice in ophthalmology and otolaryngology in the David Whitney Building.

Doctor McClelland was certificated by the American Board of Ophthalmology in 1923. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, a Fellow of the American College of Surgeons and a member of state and local medical societies.

Since 1912 he served on the staff of Grace Hospital where he was chief ophthalmologist from 1927 to 1939.

His death occurred at his home in Detroit on July 30, 1942. Surviving him are his wife, Lela French McClelland, and two daughters.

S. HANFORD McKEE

Doctor McKee was born in the Province of Quebec in Canada in 1874. He attended McGill University, graduating from the Medical Department in 1900. He was attracted early to the practice of ophthalmology and underwent his training abroad. Much time was spent in London under the masters of that day, but the most intensive study was in Freiburg under the supervision of the late Professor Axenfeld. Returning to Canada, he established himself in Montreal where more than half of his time was devoted to clinics and teaching.

Early in World War I, he was commissioned in the medical forces of the Canadian Army and soon was sent overseas. At first, he served with troops in a general medical capacity, eventually commanding a Field Hospital. Later on, he was employed in his specialty. The events subsequent to the War, depressing as they were, did not destroy his enthusiasm for his chosen field of endeavor, and he continued to work long hours as clinician, teacher and practitioner.

Doctor McKee was never a prolific writer, but what he did write was sound and thorough. His first major literary work was the translation and editing of Axenfeld's Bacteriology of the Eye, a massive tome that he considered re-writing only last year. Doctor McKee could be counted upon for two or three papers a year, papers that were based upon original laboratory investigations or upon clinical observations of the most meticulous type. What he wrote was worth reading.

"Han," by which name he was universally known, joined the Academy in 1908, but was not a regular attendant until immediately after the War. However, from then on, he hardly ever missed a meeting. He was in great demand in the Section on Instruction, both by the membership at large and the Committee of the Section. He gave cheerfully of his time and energy, and the men who attended his courses early recognized that he was a born teacher. He also belonged to the American Ophthalmological Society and the Section on Ophthalmology of the American Medical Association.

A wrinkling of his nose and a twinkling of his eyes attested to Han's well developed sense of humor. He loved a good story and if it was one that he thought would be useful, out would come the little note book and he would jot down a few cryptic key words. Charming as was his personality, he was of a retiring type, never seeking to intrude. His election to the presidency of the Academy

was met with universal favor and acclaim for he was beloved, not alone for his work, but even more for himself.

His death occurred on November 25, 1942.

RICHMOND McKINNEY

Doctor McKinney was born in Memphis, Tennessee, on June 6, 1874. He received his degree in medicine from Memphis Hospital Medical College (University of Tennessee College of Medicine) in 1895, following which he engaged in postgraduate study in otolaryngology at the University of Berlin and Christian Brothers College in Memphis. He practiced his specialty in Memphis.

Doctor McKinney was professor of otolaryngology at the University of Tennessee College of Medicine. He served on the staffs of the Baptist Memorial, Memphis General and John Gaston hospitals.

He was certificated by the American Board of Otolaryngology in 1929. He was a Life member of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association, past president and secretary of the American Broncho-Esophagological Association, member of the American Laryngological Association and the American Laryngological, Rhinological and Otological Society, and a Fellow of the American College of Surgeons.

Doctor McKinney served as president of the Memphis Country Club for 14 years.

He was author of the text book "Syllabus of Diseases of the Ear, Nose and Throat," as well as many papers concerning his specialty.

His death occurred in Memphis on June 30, 1942. Surviving are two sisters, Mrs. C. K. Lewis and Mrs. R. W. Ryden.

WILLARD GEIST MENGEL

Doctor Mengel was born in Trevorton, Pennsylvania, on August 30, 1896.

He attended Trevorton High School and Albright College, Myerstown, Pennsylvania. He obtained his medical degree from the University of Pennsylvania Medical School in 1921. Doctor Mengel was a resident physician at Coaldale Hospital, Coaldale, Pennsylvania, from 1921-1922. He engaged in postgraduate study in ophthalmology at the University of Pennsylvania Medical School in 1923.

His practice was limited to ophthalmology. He was senior assistant surgeon at Wills Hospital, Philadelphia, assistant professor of ophthalmology at the Graduate School, Pennsylvania University, and ophthalmologist on the staff of the Camden County General Hospital, Grenlock, New Jersey.

He was certificated by the American Board of Ophthalmology in 1935, and was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, Fellow of the College of Physicians of Philadelphia and a member of Camden County and New Jersey medical societies.

Doctor Mengel practiced ophthalmology in Camden, New Jersey, for almost 20 years, and he was a member of the staff of the Wills Eye Hospital, Philadelphia, for 15 years.

His death occurred in Camden on January 27, 1943. He is survived by his brother, Dr. John G. Mengel, Lebanon, Pennsylvania, and a sister, Mrs. F. E. Luckenbill, Reading, Pennsylvania.

STANLEY GEORGE MINER

Doctor Miner was born in Detroit, Michigan, on February 4, 1861. He attended public schools in Detroit and obtained his medical degree from Detroit College of Medicine (now Wayne University College of Medicine) in 1882. In 1935 the University bestowed upon him the honorary degree of Master of Medical Science. He began to practice his specialty in Detroit in 1882.

Doctor Miner was emeritus professor of otology, laryngology, rhinology and physical diagnosis at Wayne University College of Medicine. He was chief of staff of St. Mary's Hospital, Detroit.

He was a Life member of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association, the Michigan State Medical Society and the Wayne County Medical Society. He had been a member of the city public welfare commission from 1913 to 1919. Doctor Miner was influential in establishing the Detroit Receiving Hospital.

His death occurred in Detroit on June 28, 1942. He is survived by two daughters and a brother.

EDWARD POWERS NORCROSS

Doctor Norcross was born in Janesville, Wisconsin, on February 15, 1878.

He attended Beloit Academy, Beloit, Wisconsin, and the University of Wisconsin. He obtained his medical degree from Northwestern University in 1904, following which he interned at Cook County Hospital, Chicago, 1904-1905. He limited his practice to otolaryngology and served as senior member

of the staff of St. Luke's Hospital, Chicago, and attending otolaryngologist at Highland Park Hospital, Highland Park, Illinois.

Doctor Norcross was certificated by the American Board of Otolaryngology in 1925. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association and state and county medical societies. He was a Fellow of the American College of Surgeons.

In 1917-1918 he served on the Medical Advisory Board No. 10, Selective Service.

Doctor Norcross was an Episcopalian, and he was a member of the vestry of Grace-St. Luke's Chapel, Chicago.

He was a member of Chi Psi, Nu Sigma Nu and Alpha Omega Alpha fraternities.

His death occurred in Highland Park, Illinois, on August 5, 1942. His wife and three children by a former marriage survive: Emily D. Norcross, Dr. Pliny Norcross and Lieut. E. P. Norcross, Jr.

EUGENE P. NORWOOD

Doctor Norwood was born on May 27, 1877, in Delville, Texas.

His academic education was received in the Texas Agricultural and Mechanical College, Bryan. His medical education was obtained in the Medical Department of the University of Texas, Galveston, from which he was graduated in 1911. He engaged in postgraduate study at Tulane University School of Medicine and at Chicago University in preparation for his elected sepecialties of ophthalmology and otolaryngology. Doctor Norwood began the general practice of medicine at Kerens, Texas, where he remained for several years. After specializing, he located in Corsicana, Texas, in 1923, where he practiced his specialties until February, 1942. He then located at Temple, Texas, where he was in charge of the eye, ear, nose and throat department of the King's Daughters Hospital until his death.

Doctor Norwood was certificated by the American Board of Otolaryngology in 1938. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association and state and county medical societies. He was a past president of the Navarro County Medical Society. He was also a member of the Central Texas District Society and the Dallas Academy of Ophthalmology and Otolaryngology. During his practice at Corsicana, he served as eye, ear, nose and throat specialist to the State Orphans' Home for 14 years. Doctor Norwood served as Captain in the Medical Corps of the British Army as Regimental Surgeon during World War I, and after the Armistice, in a British hospital in the Army of Occupation in Cologne.

Doctor Norwood was a member of the Episcopal Church and a 32nd Degree Mason. He was a member of the American Legion. He enjoyed the highest esteem of his medical associates and the respect and admiration of all with whom he came in contact as a physician and citizen.

His death occurred on January 1, 1943, in Temple, Texas. He is survived by his wife and a daughter, Mrs. J. E. Pennington, Jr., of Dallas.

SAMUEL POINDEXTER OAST

Doctor Oast was born at Portsmouth, Virginia, on November 27, 1886.

His undergraduate education was received at Virginia Polytechnic Institute and the University of Virginia. His medical school was the University of Pennsylvania from which he was graduated in 1911. He served his internship at Pennsylvania University Hospital from 1911 to 1913 and was a resident at the New York Eye and Ear Infirmary from 1920 to 1923 where he served first in aural and then in ophthalmic departments. Following this period of residency he was made in 1925 an assistant surgeon, in 1937 a full surgeon and in 1941 an honorary surgeon. In this latter capacity he served until his death. In addition he was assistant attending surgeon at Bellevue Hospital, assistant clinical professor of ophthalmology at New York University and consulting ophthalmic surgeon at the U. S. Marine Hospital, Ellis Island.

Doctor Oast was certificated by the American Board of Ophthalmology in 1926. He was an active member in the following organizations: American Academy of Ophthalmology and Otolaryngology, American Medical Association, American College of Surgeons, American Ophthalmological Society, Academy of Medicine of New York and the New York Ophthalmological Society.

During the first World War Doctor Oast entered the Ambulance Corps of the American Hospital in Paris from the Medical Reserve Corps of the United States Army in which he had enlisted in 1916. He later became affiliated with the American Expeditionary Army with which he saw active fighting service. After the armistice until August 15, 1919, he was demobolized at Camp Lee, Virginia.

His death occurred in Portsmouth, Virginia, on October 26, 1942. Surviving Doctor Oast are his wife, the former Dorothy Francis Harper, two sons, Samuel P. Oast, 3rd, and Robert Waring Oast, his parents, one sister and six brothers.

LUTHER CROUSE PETER

Doctor Peter, the son of Rev. Jacob and Harriet Jane (Crouse) Peter, was born in St. Clairsville, Pennsylvania, February 14, 1869. He received his elementary education in the schools at Manheim, Pennsylvania, attended Susquehanna University until 1888, and was graduate from Gettysburg College in 1891, where he did post-graduate work until 1892, at which time he received his M.A. degree. He then entered the University of Pennsylvania, and was graduated with the degree of Doctor of Medicine in 1894. In 1926 Gettysburg College awarded him a Doctor of Science degree, and Susquehanna University awarded him a similar degree in 1934; in 1938 he received a Doctor of Laws degree from Gettysburg College. The Phi Beta Kappa Chapter of Susquehanna University elected him to post-graduate membership in 1925.

He was married to Carrie Chrystine Moser, who survives him, on June 20, 1916. They had no children.

He was appointed assistant professor of ophthalmology at the Philadelphia Polyclinic Hospital and School for Graduate Medicine in 1910, and associate professor of ophthalmology at the same institution, in 1918, which was at that time absorbed by the University of Pennsylvania as the Graduate School of Medicine of the University, and finally, in 1919, he became professor of ophthalmology. In 1930 he resigned from the faculty of Temple University as professor of ophthalmology, having served in that capacity since 1917.

He was a member of the American Board of Ophthalmology from 1929 to 1937, its president 1935 to 1937; secretary of the American Academy of Ophthalmology and Otolaryngology from 1919 to 1926, and president of that Academy 1928-1929; elected to the American Ophthalmological Society in 1922, to the Philadelphia College of Physicians in 1917, and to Fellowship in the American College of Surgeons in 1920. In 1922, Doctor Peter was secretary of the International Congress of Ophthalmology held in Washington, D. C.; was guest lecturer at the Oxford Congress (England) in July, 1920, his subject being "Perimetry," and again in July of 1932, this time his subject being "The Treatment of Non- Paralytic Squint."

Many articles were contributed by him to ophthalmologic literature in perimetry, disturbances of the extra-ocular muscles, in neuro-ophthalmology, the surgery of cataract and of glaucoma and more recently in the treatment of retinal separation. His book "The Principles and Practice of Perimetry" appeared in four editions, and in 1940 "The Extra-Ocular Muscles" had its third edition. While actively engaged in practice, he was on the hospital staffs, as ophthalmologist, of The Graduate Hospital of the University of Pennsylvania, Rush Hospital for Consumption and Allied Diseases, Friend's Hospital for Mental and Nervous Diseases and the Roxborough Memorial Hospital.

Doctor Peter's death occurred in Philadelphia, on November 12, 1942.

WILLIAM AUBREY POOLE

Doctor Poole was born in Poole, Kentucky, on September 19, 1881.

He attended the University of Kentucky before enrolling at St. Louis University where he obtained his medical degree in 1906. Doctor Poole practiced general medicine in Henderson, Kentucky, following which he engaged in post-graduate study in ophthalmology and otolaryngology at the New York Post-Graduate School, Chicago Eye and Ear Infirmary, Washington University and Tulane University School of Medicine. In 1918 he began to practice his specialties in Lexington where he served on the staff of the Good Samaritan Hospital and City Hospital. He was a member of the staffs of Letcher Hospital (Henderson, Ky.), St. Mary's Hospital (La Salle, Ill.), St. Margaret's Hospital (Spring Valley, Ill.), and Harris Hospital (Mendota, Ill.). He was ophthalmologist to the U. S. Employees Compensation Commission and at one time health officer of Henderson County.

Doctor Poole was certificated by the American Board of Otolaryngology in 1936. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the Southern Medical Association, Fayette County Medical Society and past president of the Henderson County Medical Society.

During World War I he was a Major in the medical corps of the United States Army.

Doctor Poole was active in Masonic Orders: Shrine, Knights Templar, and Washington Council. He was a member of the American Legion, Lions Club and Y.M.C.A. Writing and music were his hobbies.

His death occurred on June 2, 1942, in Lexington, Kentucky, as a result of injuries received in an automobile accident in March, 1942. Surviving are his wife, Ann Porter Poole, a son, Cyrus A. Poole and a daughter, Elizabeth Poole Arnold.

HOWARD THOMAS ROBINSON

Doctor Robinson was born on May 23, 1880, in Harford County, Maryland.

He attended the public schools in Harford and received his medical degree from the University of Maryland, Baltimore, in 1904. He began practice then in Grantsville, Maryland, where he remained nine years. Doctor Robinson later engaged in post-graduate study in ophthalmology and otolaryngology at the New York Post-Graduate Medical school. He came to Cumberland, Maryland, as an eye, ear, nose and throat specialist in 1915.

Doctor Robinson was on the courtesy staff of the Allegany Hospital of the Sisters of Charity and a member of the staff of the Memorial Hospital.

He was a Senior member of the American Academy of Ophthalmology and Otolaryngology, a member of the American Medical Association, the Allegany-Garrett County Medical Society, the Medical and Chirurgical Faculty of Maryland and the Rush Medical Society.

Fraternally, Doctor Robinson was a 32nd Degree Mason.

His death occurred in Cumberland, Maryland, on December 3, 1942. He is survived by his wife and two sons, Howard T. Robinson, Jr., and William H. Robinson, both of whom are students at Johns Hopkins University, Baltimore.

JOSEPH MORRIS ROSENTHAL

Doctor Rosenthal was born on March 17, 1885, in Odessa, Russia.

He came to the United States from Germany when he was eight years old and made his home in Philadelphia. He received his degree in medicine from Jefferson Medical College in 1910 and from 1911-1912 interned at Jewish Hospital, Philadelphia. Doctor Rosenthal engaged in postgraduate study in ophthalmology at the New York Eye and Ear Infirmary following which he returned to Philadelphia to practice his specialty. From 1917 to 1930 he served as assistant ophthalmologist on the staff of Jewish Hospital; from 1930 to 1934 he served as associate ophthalmologist, and in 1934 he was made senior attending ophthalmologist. He was a former instructor in ophthalmology at Jefferson Medical College.

Doctor Rosenthal was certificated by the American Board of Ophthalmology in 1938. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association and state and local medical societies.

During World War I Doctor Rosenthal was commissioned a Lieutenant in the Medical Corps of the U. S. Navy.

His death occurred on September 3, 1941. He is survived by his wife.

GEORGE SAMUEL ROW

Doctor Row was born on November 1, 1866, in Osgood, Indiana.

He was graduated from Miami College of Medicine, Cincinnati, in 1890. Immediately afterwards Doctor Row became affiliated with Doctor Ayres and Doctor Sattler in Cincinnati and remained with them six years. This was the first clinic of ophthalmology founded in the Middle West. He moved to Indianapolis in 1896 where he practiced ophthalmology until his death.

Doctor Row had been clinical professor in ophthalmology at the old Central College of Medicine and Surgery and associate professor in ophthalmology at Indiana University School of Medicine. He was a member of the staffs of City, Methodist and St. Vincent's hospitals, Indianapolis.

In 1911 Doctor Row was elected to membership in the American Academy of Ophthalmology and Otolaryngology. He was a member of the American Medical Association and state and local medical societies.

Golf, fishing and hunting were his hobbies.

His death occurred on July 8, 1942, in Indianapolis. Surviving him are his wife, Mary Hamilton Row, a daughter, Margaret Row Walbridge, and a son, Dr. D. Hamilton Row.

ABRAHAM NOAH SCHILLER

Doctor Schiller was born in New York City in 1886.

He attended the College of the City of New York, and received his medical degree in 1907 from Columbia University College of Physicians and Surgeons, New York, following which he engaged in post-graduate study in ear, nose and throat in Vienna. He began the practice of his specialty in New York City in 1909.

Doctor Schiller was attending consultant in ear, nose and throat at Jewish Memorial Hospital and consultant in ear, nose and throat at City Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the New York Academy of Medicine and state and county medical societies.

He was founder of the Jewish Memorial Hospital and past president and honorary

president of the medical board of the Jewish Memorial Hospital.

His interests other than medical were in travel.

His death occurred in Miami Beach, Florida, on December 17, 1942. His wife, Jenny, and daughter, Ellain, survive him.

LOUIS JOSEPH SEBERT

Doctor Sebert was born in Whitby, Ontario, Canada, in 1886.

He received his early education at Brooklin Public School and Whitby Collegiate, and he was graduated from the faculty of medicine, University of Toronto, in 1911. He served as interne at St. Michael's Hospital, Toronto, from 1911 to 1912 and engaged in post-graduate study in ophthalmology in New York and Vienna. Following his return to Canada he was appointed to the staff of St. Michael's Hospital, where he later became chief of the department of ophthalmology. He was senior demonstrator in ophthalmology at the University of Toronto. A consultant at St. Joseph's Hospital since it was founded in 1921, Doctor Sebert was for some years on the staff of the Hospital for Sick Children. He was a member of the Medical Advisory Board of St. Michael's Hospital.

Doctor Sebert was certificated by the American Board of Ophthalmology in 1924. He was a member of the American Academy of Ophthalmology and Otolaryngology, the Canadian Medical Association and the Canadian Ophthalmological Society.

While attending University, Doctor Sebert starred in track athletics. In 1908 he represented Canada at the Olympic games in London, England.

In 1928 he was the Separate School Board representative on the Toronto Board of Education.

He was a member of Our Lady of Sorrows Roman Catholic Church, the Knights of Columbus, Kiwanis Club, Lakeview Golf Club, Toronto Stamp Collectors' Club and the Athletic Board of the University of Toronto.

Doctor Sebert's death occurred on December 3, 1942, at St. Michael's Hospital, Toronto. Surviving are his wife, Lillian Mason Sebert; three sons, James, John and Lieutenant Louis Sebert, a daughter, Mary, and his mother.

FREDERICK CASIMIR SIMON

Doctor Simon was born on January 7, 1875, in St. Louis, Missouri.

He attended public schools in St. Louis and St. Louis College of Pharmacy. He obtained his medical degree from Missouri Medical College (Washington University, St. Louis) in 1899 and interned at St. Louis City Hospital. Doctor Simon engaged in post-graduate study in ear, nose and throat at the University of Berlin and in Vienna. He began to practice his specialty in St. Louis in 1900.

Doctor Simon was assistant to the chief surgeon of the St. Louis Transit Company, he served on the staff of the ear, nose and throat clinic of Washington University for a period of seven years and at one time was an instructor in otolaryngology at St. Louis University School of Medicine.

He was certificated by the American Board of Otolaryngology in 1929 and was elected to membership in the American Academy of Ophthalmology and Otolaryngology the same year. He was a member of the American Medical Association, Fellow of the American College of Surgeons, member of the Missouri State Medical Society, the St. Louis Medical Society and the St. Louis Ear, Nose and Throat Club. Doctor Simon was a past president of the Alumni Association of Washington University Medical School and a member of Nu Sigma Nu medical fraternity.

During World War I he was appointed to the Physical Examination Board in St. Louis and in addition served on the examining board and as recruiting officer for the Aviation Signal Corps Section. He also served on the staff at the Post Hospital, Love Field, Dallas, Texas.

Doctor Simon's death occurred in St. Louis, Missouri, on December 31, 1942. He is survived by his wife and a daughter, Virginia.

HARRY BAYLISS STOKES

Doctor Stokes was born in India in 1904, and he moved to London, England, with his parents a few years later. There he received his early education, coming to the United States at the age of 20. He matriculated at the University of Michigan Medical School, from which he was graduated in 1929. Following internship, Doctor Stokes did special work in otolaryngology at Bellevue Hospital, New York City, and was certificated by the American Board of Otolaryngology in 1933. He practiced in Rochester, New York, and in Tucson, Arizona, for short periods of time, moving to Omaha in 1936. For the past six years he was an associate in the office of Drs. Gifford, Stokes and Stokes.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the Omaha Mid-West Clinical Society and state and local medical associations. He had a very ac-

tive practice in the midst of which he found time to carry through his assignments as instructor in otolaryngology at the University of Nebraska College of Medicine.

Doctor Stokes was a Major in the medical corps of the United States Army and was killed in action on January 2, 1943, while on duty in Africa.

He is survived by his wife, two daughters, Suzan and Shelley, and one brother, Dr. William H. Stokes, of Omaha.

JAMES CRAIG STRONG

Doctor Strong was born in Paintsville, Kentucky, on March 31, 1878, the son of Attorney Thomas B. and Nancy Strong.

He attended public schools in West Virginia, the West Virginia State Normal School and the University of Nashville. He obtained his degree in medicine from the Kentucky School of Medicine in 1903, following which he engaged in post-graduate study in ophthalmology and otolaryngology at the Chicago Eye, Ear, Nose and Throat College and at the Polyclinic, in New Orleans. Doctor Strong practiced in Newcastle, Arkansas; Forest City, Arkansas, and Dillon, Colorado, before moving to Leadville, Colorado, where he remained until his death. He was a member of the staff of St. Vincent Hospital, Leadville.

Doctor Strong was a Senior member of the American Academy of Ophthalmolgy and Otolaryngology, a member of the American Medical Association, the Colorado Medical Society, the Colorado Ophthalmological Society and past president and for 20 years secretary of the Lake County Medical Society.

He was deeply interested in lodge work and was past noble grand master of the I.O.O.F., an Order of Eastern Star patron, a member of A. F. and A. M. Royal Arch Masons and the Rebekahs. He was a member of the Leadville Chamber of Commerce and the Leadville Library Association Board.

His death occurred on October 14, 1942, in Leadville, Colorado. He is survived by his wife, Nannie B. Strong, and four sons, all of whom are in the service of their country — Dr. Derrell R., Dr. James Maitland, Ensign George Ralph and Dr. James Craig, Jr.

EDWARD FRANKLIN STROUD

Doctor Stroud was born on May 25, 1884, in Waxahachie, Texas.

He attended private school in Midlothian, Texas, and Trinity University in Waxahachie, Texas. He received his medical degree from Tulane University Medical School in 1908, following which he practiced general medicine in Tulsa, Oklahoma. From 1913 to 1915 Doctor Stroud engaged in post-graduate study in ear, nose and throat at the New York Post-Graduate Medical School, serving as assistant and house surgeon following his residency there. He practiced his specialty in Dallas, Texas, four years, serving on the staff of St. Paul's Hospital. He then moved to Corpus Christi, Texas, where he became attending otolaryngologist at the Fred Roberts Memorial Hospital, a member of the staff of Medical Professional Hospital and at one time chief of staff of Spohn Hospital.

Doctor Stroud was certificated by the American Board of Otolaryngology in 1926. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, Fellow of the American College of Surgeons, member of the Texas Ophthalmological and Otolaryngological Society and past president of Nueces County Medical Society.

During World War I Doctor Stroud was a Captain in the United States Army Medical Corps and he served in France. He was chief of the ear, nose and throat section of Base Hospital, Le Mons, France, 1918-1919.

His death occurred in Corpus Christi, Texas, on December 31, 1942. His wife, Ethel A. Stroud, a daughter and a son survive.

FRANK VINSONHALER

Doctor Vinsonhaler was born in Graham, Missouri, on April 14, 1864.

He attended Northwestern Normal School, Oregon, Missouri, and later studied medicine at the College of Physicians and Surgeons, Columbia University, New York, from which he received his degree in medicine in 1885.

Doctor Vinsonhaler practiced medicine in the Black Hills region six years, following which he engaged in post-graduate study in ophthalmology and otolaryngology at the University of Vienna and at the Heidelberg, in Germany. He spent a year at the Royal Ophthalmic Hospital in England.

Doctor Vinsonhaler came to Little Rock, Arkansas, from the old Missouri Medical College, now St. Louis University, in 1893, to accept a position as professor of ophthalmology, otology, rhinology and laryngology at the University of Arkansas Medical School. When he joined the faculty of the University, the late Dr. James Dibrell was dean and professor of anatomy. Doctor Vinsonhaler served on the faculty continuously, except for two years during World War I, until his retirement in 1938. He continued private prac-

tice as eye, ear, nose and throat specialist until 1927, when he was elected dean of the Medical School which position he held until his retirement. From 1932 to 1939 he was professor of medical history and ethics at the University of Arkansas Medical School.

Under his guidance the Medical School was moved from a small building at Second and Sherman Streets, with virtually no clinical facilities, into a new $500,000 building housing more than $150,000 worth of the latest type of equipment and an enormous clinic.

A Life member of the American Academy of Ophthalmology and Otolaryngology, Doctor Vinsonhaler was certificated by the American Board of Ophthalmology in 1920. He was a member of the American Medical Association, a charter member of the American College of Surgeons, and member and past president of the Arkansas Medical Society and the Pulaski County Medical Society.

Doctor Vinsonhaler was an active 33rd Degree Mason and had served as sovereign grand inspector general of the Supreme Council of the Scottish Rite Jurisdiction of Arkansas for several years.

During the Spanish-American War he was a member of the governor's staff, and during World War I he was Commander of Base Hospital 109, Vichy, France. He retired from the army at the close of the war with the rank of Colonel.

In 1935, along with six others, including Lincoln Ellsworth, the explorer, Doctor Vinsonhaler was awarded a distinguished alumnus medal at Columbia University by Dr. Nicholas Murray Butler, bearing the following citation: "Distinguished ophthalmologist and dean of Medical School, University of Arkansas; loyal and devoted alumnus to this university."

Doctor Vinsonhaler was an active civic worker and helped to organize the Community Chest early in the 1920's. He served as president of the organization in 1925 and 1926.

He was an honorary member of Phi Beta Kappa fraternity. He helped to organize and served as first commander of the M. M. Eberts American Legion Post. He was a member of the Rhodes Scholarship Selection Committee for Arkansas.

He acted as Belgian consul for Arkansas for many years.

Doctor Vinsonhaler's death occurred on September 1, 1942. He is survived by two daughters, Mrs. Mariane McCain, Scarsdale, New York, wife of Arthur W. McCain, vice president of Chase National Bank, New York City, and Mrs. Frances Williams, Little Rock, wife of Captain B. B. Williams; a son, George Vinsonhaler, general agent of the John Hancock Insurance Company at Cincinnati, Ohio; two brothers, Judge D. M. Vinsonhaler, Omaha, Nebraska, and Harry Vinsonhaler, St. Louis, Missouri.

CARL B. WAGNER

Doctor Wagner was born on May 8, 1876, in Hohenzollern, town of Hechingen, Germany.

From 1894 to 1897 he assisted and studied with Dr. Lucien Howe, internationally known specialist in ophthalmology, in Buffalo, New York. From 1897 to 1905 Doctor Wagner did research work in Detroit, Michigan. In 1909 he received his medical degree from the Illinois Medical College. He specialized in ophthalmology and otolaryngology.

He was formerly clinical professor of ophthalmology at Loyola University, member of the consulting staff of Cook County Hospital and served on the staff of the Chicago Eye, Ear, Nose and Throat Hospital.

Doctor Wagner was elected to Honorary Life membership in the American Academy of Ophthalmology and Otolaryngology in 1912; he was a member of the American Medical Association, the International Congress of Ophthalmology, the American Association for the Advancement of Science, the Illinois State Medical Society, the Chicago Ophthalmological Society and the Chicago Medical Society.

He was the originator of the Wagner Malinger Ear Phone and Eye Charts, and he wrote many articles concerning ocular nystagmus.

His death occurred on February 1, 1943, in Chicago. He is survived by his wife, Willa W. Wagner.

C. B. WALKER

Doctor Walker was born in 1884.

He received his degree in medicine from Johns Hopkins University School of Medicine, Baltimore, in 1911. He was clinical professor of surgery (ophthalmology) at the University of Southern California School of Medicine, Los Angeles, formerly professor of clinical ophthalmology at the College of Medical Evangelists, Los Angeles, and at one time assistant in ophthalmology at the Harvard Medical School.

Doctor Walker was certificated by the American Board of Ophthalmology in 1931, and he served as a member of the Board. He was a member of the American Academy of Ophthalmology and Otolaryngology, The

American Ophthalmological Society, Association for Research in Ophthalmology, Inc., Western Ophthalmological Society and the Pacific Coast Oto-Ophthalmological Society. He was a past president of the Los Angeles Society of Ophthalmology and Otolaryngology.

Doctor Walker served on the staff of the California Hospital, Eye and Ear Hospital, Good Hope Clinic, Los Angeles County Hospital and the Hospital of the Good Samaritan. He had formerly served on the staff of Peter Bent Brigham Hospital, Boston.

In 1914 he received the first Knapp medal from the Section on Ophthalmology of the American Medical Association for his paper on "Topical Diagnosis of the Hemiopic Pupillary Reaction and the Willbrand Hemianopic Prism Phenomenon, with a New Method of Performing the Latter."

His death occurred on July 3, 1943.

NELSON SHELLY WEINBERGER

Doctor Weinberger was born in Spinnerstown, Pennsylvania, on June 27, 1882.

He attended Perkiomen Seminary, Pennsburg, Pennsylvania, and Westchester Normal School, Pennsylvania. He received his medical degree from the University of Pennsylvania Medical School in 1905, and began to practice general medicine in Quakerstown, Pennsylvania, in 1905. In 1909 Doctor Weinberger engaged in post-graduate study in ophthalmology and otolaryngology at Wills Eye Institute and the University of Pennsylvania. From 1912 until his death he was chief of the department of ophthalmology and otolaryngology at the Robert Packer Hospital, Philadelphia, and the Guthrie Clinic, Sayre, Pennsylvania.

He was certificated by the American Board of Otolaryngology in 1925. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, Fellow of the American College of Surgeons, member and formerly vice president of the American Laryngological, Rhinological and Otological Society, member of the Association for Research in Ophthalmology, Pennsylvania State Medical Society, Bradford County Medical Society and N. Y. and N. E. Association of R. R. Surgeons.

Dr. Weinberger's death occurred on June 28, 1942. He is survived by his wife, Martine R. Weinberger, and a son, John.

WALTER JOSEPH WHERRY

Dr. Walter Wherry, son of the late Dr. William P. Wherry, was born in Omaha, Nebraska, on September 19, 1905.

He attended Omaha grade schools, the Onarga Military Academy at Onarga, Illinois, and received his medical degree from the University of Nebraska in 1935. He served his interneship from 1935 to 1936 at St. Vincent's Hospital, Portland, Oregon, and he was a resident in otolaryngology at the Massachusetts Eye and Ear Infirmary from 1936 to 1939. In 1939 he returned to Omaha where he practiced his specialty with his father.

Doctor Wherry was certificated by the American Board of Otolaryngology in 1941 and was made a member of the board. He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the Omaha-Douglas County Medical Society, the Nebraska State Medical Association, the Midwest Clinical Society and the Omaha-Council Bluffs Eye, Ear, Nose and Throat Society.

He was a member of the staffs of Nebraska University Hospital and St. Catherine's Hospital and was a member of the visiting staff of Bishop Clarkson Memorial Hospital. He was assistant professor of otolaryngology at the University of Nebraska Medical College.

Doctor Wherry was a member of the Kiwanis Club, the Omaha Athletic Club, the Omaha Club and the Field Club. His hobbies were photography, fly fishing and golf. His latest hobby was building a scale model railway station.

His death occurred on April 1, 1943, in Omaha. Surviving are his wife, mother, two daughters, a sister and a brother.

American Association of Eye, Ear, Nose, and Throat Society Secretaries

WALTER H. THEOBALD, M.D.
President

KENNETH L. CRAFT, M.D.
Secretary-Treasurer

The fourth annual meeting of the American Association of Eye, Ear, Nose and Throat Society Secretaries convened October 12, 1942, at the Palmer House, Chicago. The meeting was held at the time of a luncheon provided through the courtesy of the American Academy of Ophthalmology and Otolaryngology, and was called to order by Dr. Walter H. Theobald, President of the Association. The roll was called and annual dues of $2.00 were collected from each of the 27 members present.

Doctor Theobald introduced Dr. John L. Myers, Secretary for Otolaryngology of the Academy, who spoke as follows:

"You secretaries are so situated that you can be of help to the Academy; you can also be of service to each member of your respective societies. A few years ago I suggested that each local society select a secretary and keep that same man in official position for several years. By so doing, each secretary becomes acquainted with the group, such as we have here today, and all of you collectively become familiar with the function of the Academy—this makes for more efficient work on your part, both for the Academy and for your local societies.

"One way by which you can help the Academy is for each of you to familiarize yourself with the talent in your local organization. When you observe a man (1) doing some unusual research work, (2) preparing a paper of special interest or (3) making a good motion picture, be sure to give that information to Doctor Benedict. All this information can then be tabulated and considered by the Program Committee of the Academy in making up scientific, motion picture and instruction course programs for the annual meetings.

"Here is another suggestion. Some of you say you have trouble in developing programs for your local societies. One thing you should keep foremost in your organization is to develop your local talent. This can be done by encouraging members to select some subject and study it until they are familiar with every phase of that subject. Encourage the reporting of cases by kodachrome photography. If various members do this, in time a great number of interesting case reports will be accumulated, and an interesting and instructive evening can be spent in going over all these studies. Pictures of the patient when first seen, a brief history, laboratory findings, x-ray findings, operative procedure, pathology, a microscopic picture of the pathology present, a fever chart and other charts, a picture of the patient at various stages of convalescence, when dismissed and later on if so desired, are data to be accumulated in making a report concerning an interesting case.

"Then each local society can draw on the Academy for material for its programs. I am sure you have seen the motion pictures with sound which show

the ear from otoscopy through to caring for thrombosis of the lateral sinus. These motion pictures can be secured by each society. They are interesting and instructive. As the years go by, there will be many more sound motion pictures. Even now you can get a motion picture on almost any subject you wish to consider.

"Members of each local society can be helped by having their members who are members of the Academy report on courses taken at annual meetings of the Academy. By doing this, the information imparted in these refresher courses by America's best teachers is being disseminated all over the country and made to reach many thousands who cannot attend every annual meeting. This will make for better work in your local societies and create a desire in many to attend the Academy meetings where they can come in personal touch with and be inspired by the men who give these courses.

"The Academy is more and more becoming a *Teaching Institution*. If you secretaries will earnestly endeavor to carry out the lines of work herein outlined, you will be helping the Academy on the one hand and on the other, you will be disseminating knowledge accumulated by the Academy. You face a challenge and an opportunity. I thank you."

Following this inspiring talk by Doctor Myers, there was discussion concerning the question of whether or not the American Academy of Ophthalmology and Otolaryngology would meet in 1943, due to war conditions. It was moved by Dr. Edley H. Jones, Vicksburg, Mississippi, and seconded by Dr. Vernon M. Leach, Chicago, Illinois, and passed, that this Association recommend that the Academy hold its regular meeting in 1943. This resolution was imparted to the Council of the Academy by Dr. Walter H. Theobald.

The regular program of this meeting consisted of discussions by various members on "Program Features of Especial Value in E.E.N. & T. Society Meetings." The discussion was opened by Dr. W. E. Keith, Kansas City, Missouri, Secretary of the Kansas City Society of Ophthalmology and Oto-Laryngology, who spoke as follows:

"This year for the first time we are trying out the idea of a review of certain courses taken by our members attending the convention of the American Academy of Ophthalmology and Otolaryngology, and the initial attempt will be made by five members at our November 19th meeting.

"We have tried out the broadcasting idea, but it was in conjunction with the local medical society and for some reason did not provoke sufficient interest.

"One meeting which produced a big attendance with plenty of favorable comment was one with motion pictures in color from various authors (loaned directly), such as Arthur Bedell, Pierre Viole, Conrad Berens, Edmund Spaeth, Edmund Fowler, Eugene Dalton, Louis Clerf, O. E. Van Alyea and Paul Holinger. Each film was presented by a local member who had reviewed the reel and so was prepared to show it.

"Our members are given the opportunity to describe by technic demonstrations and slides such data as displacement therapy with patient's use of iodine in OORL, some interesting observations in the use of polarized light in refraction, kodachrome slides taken personally, etc.

"Our final meeting of the year is comprised of clinics pre-arranged by especially chosen staffs (rotating) and golf with Ladies' Night at which photographs in colors are shown of all past presidents, deceased or alive. In many instances this produces much fun—for instance, when mine was taken I weighed only 134 pounds.

"Heretofore nine bulletins were published a year, but this year only four will be published. The cost for the eight pages is about $25.00, plus postage and the cost of envelopes. It is most difficult at this time to obtain articles from our Honorary Members. So far, out of 36 letters written I have received two articles."

Dr. Thomas F. Furlong, Jr., Secretary of the Philadelphia Laryngological Society, Ardmore, Pennsylvania, spoke as follows:

"Our society was founded in 1885, and for approximately the past 25 years we have had a monthly bulletin, a sample of which I have here with me. It contains the program, roster of officers, necrology, notice of election of new members, etc. One of the most important items which is contained in every bulletin is a standing invitation to all physicians, internes and medical students to attend our meetings. The meeting in May of each year is turned over to the finishing class in Otolaryngology of the Graduate School of Medicine of the University of Pennsylvania. These students sponsor the entire meeting and always put on an interesting program.

"Because of the gasoline rationing which is daily becoming more of a problem in Philadelphia, we seriously considered combining our meetings with those of our Section of Otolaryngology of the College of Physicians. This has been done, and we will continue to hold joint meetings for the duration of the war.

"Regarding the boards, ever since the American Board of Otolaryngology was founded in 1925, our society has required the certificate of this board as a prerequisite to membership."

Dr. J. E. Landis, President of the Eastern Pennsylvania Association of Eye, Ear, Nose and Throat Physicians, Reading, Pennsylvania, spoke as follows:

"It has been my observation that our specialties of ophthalmology and otolaryngology have been disunited in the smaller communities. In the larger cities, organizations have been formed, and they now serve in a very beneficial capacity to the men practicing there and in the outlying districts. In the smaller towns, where there may be anywhere from 10 to 20 specialists, there has been no unity of purpose, and the individual has become a lone player. For that reason, I believe that organization and the formation of smaller societies is beneficial from a sociability standpoint as well as from the standpoint of keeping the individual abreast of modern developments in medicine. He has opportunities of meeting speakers who excel in their field, and he would not have such an opportunity if such an organization did not exist. One may think that the local medical societies would afford him this opportunity, but unfortunately, the subjects discussed at these meetings are too general. Since our specialties are developing by leaps and bounds, due to the influence of the Academy, it is necessary that specialists in ophthalmology and otolaryngology have some direct contact with the Academy's progress. Nothing can afford this better than small organizations of this kind. I feel that these organizations should be associated with a sectional or state organization as a parent association in the development and guidance of their activities. This in turn becomes a child of the Academy, and the Academy is the so-called godfather. In this way, through these three sets of organizations, there will be developed two of the most exacting specialties in our profession and will tend to encourage the men to do better work.

"The second phase of equal importance is the association with other fellow practitioners. Many pitfalls and jealousies develop among men practicing in the same community, which, in

the majority of cases, is due only to misunderstandings and lack of knowing each other better. Local societies tend to eliminate this to a great degree. Societies encourage the reporting of unusual cases and the filing of these reports; also, the incentive for writing papers and the encouragement of postgraduate study and research are augmented.

"In reference to a program, our society meets once a year with the local dental association for the general discussion of facial maxillary conditions and associated complications from dental origin, so that we may better understand their problems and in return they understand ours. We suggest that papers be read by the members of the dental association. Once a year, we make a pilgrimage to a medical center for dry and wet clinics. Once a year, we have a social gathering. The Reading Eye, Ear, Nose and Throat Society is sponsoring radio broadcasts on general topics to the layman, explaining the dangers and the difficulties that people may encounter by neglecting what they think is a seemingly unimportant condition and hoping that they will outgrow it. The subjects that we will discuss for 15 minute periods for 13 weeks are as follows:

1. The Eyes in Air Raids.
2. Colds and Their Results.
3. Hay Fever and Conditions Like It.
4. What Is Sinusitis?
5. The Tonsil Question.
6. Deafness and Hearing Aids.
7. Hoarseness.
8. The Management of Cross Eye.
9. Eye Irritations.
10. Eye Hygiene.
11. Cataracts.
12. Glaucoma.
13. Running Ears.

"After the broadcast, these articles will be featured in the local newspapers. We encourage making photographs of unusual cases.

"In this way, let me stress the growing importance of uniting the men in small communities. Medical legislation and changes that are brought about by that can be greatly influenced and guided by our organization; by ourselves, individually, none of us can accomplish as much."

Dr. Julius W. McCall, Secretary of the Cleveland Otolaryngological Club, Cleveland, Ohio, gave the following discussion:

"The Cleveland Otolaryngological Club usually plans five meetings a year, beginning in October and ending in April. For four of the meetings, out-of-town speakers are invited. The final April meeting is reserved for the home talent.

"We have an afternoon session which is held at the Anatomical Department of Western Reserve University, through the courtesy and cooperation of Dr. Norman L. Hoerr, Professor of Anatomy. The members of the club usually demonstrate some phase of new operative surgery along with interesting features of the head and neck. Following these demonstrations the various members of the club are allowed five minutes to present interesting cases and case histories.

"We then adjourn for dinner. After the dinner the residents of the various hospitals in Cleveland present papers on some otolaryngological subject of their own choice. The out-of-town guests cast a vote for the resident's paper which they believe is the best, and a prize is awarded for it.

"Another feature of the Cleveland Otolaryngological Club program is a course of three months in anatomy of the head and neck and otolaryngological cadaver surgery which is given to the assistant residents and residents of the various hospitals of the city. The hospital pays the necessary charge for cadaver material, considering it a part of the basic training for their staff men."

Dr. Sheldon B. Freeman, Secretary of the Buffalo Ophthalmologic Club, Buffalo, New York, was next on the program:

"Mr. President, I wish to bring to the attention of the secretaries a subject which is of importance at this particular time. That is, what are the various clubs going to do about their members who are in the Military Service and have had to discontinue private practice? To save time I will tell you about the action that the Buffalo Ophthalmologic Club has taken in regard to this matter. First, those who are in the Military Service will not pay dues, and they will continue as members in good standing in the club. Second, the regular notices of the club and a report of all meetings will be forwarded to them. Third, it has been agreed that when members of our club treat patients who are known to be patients of the members in the Military Service, 50 per cent of the fee will be returned to the member in the Service. This is purely a club affair and the opticians do not take part in this third undertaking in any way. I should be very glad to hear what other clubs are doing or contemplating along this line."

Dr. Thomas F. Furlong, Jr., Secretary of the Philadelphia Laryngological Society, replied to Doctor Freeman as follows:

"In reply to Doctor Freeman, I can say with pleasure that shortly after the beginning of the war, our society passed a motion to the effect that any member, taking care of a patient of a doctor from the Philadelphia area who has gone into Service, would see to it that 50 per cent of such a fee or fees collected would be given to the doctor on active duty. I may also say with pleasure, our Section of Otolaryngology of the College of Physicians of Philadelphia, of which many of us are also members, passed the same motion."

Dr. Kenneth L. Craft, Indianapolis, Indiana, Secretary of the Indianapolis Ophthalmological and Otolaryngological Society, mentioned that the Indianapolis society had adopted essentially the same plan.

Election of officers and directors for the coming year was as follows:

President .....Dr. W. H. Morrison
Omaha, Nebraska

Secretary-Treasurer....Dr. Kenneth L. Craft
Indianapolis, Indiana

Member of the
Executive Council...Dr. W. H. Theobald
Chicago, Illinois

Directors:

District No. 1.....Dr. R. T. Paton
New York, N. Y.

District No. 2.....Dr. S. B. Freeman
Buffalo, N. Y.

District No. 3.....Dr. L. A. Brown
Atlanta, Georgia

District No. 4.....Dr. Sam H. Sanders
Memphis, Tennessee

District No. 5.....Dr. Daniel S. DeStio
Pittsburgh, Pennsylvania

District No. 6.....Dr. R. T. Rank
Milwaukee, Wisconsin

District No. 7.....Dr. W. E. Keith
Kansas City, Missouri

District No. 8.....Dr. J. C. Ogden
Long Beach, California

Following the election, Doctor Theobald, retiring President, turned the meeting over to Doctor Morrison. No further business was transacted, and the meeting was adjourned at 2 p.m.

KENNETH L. CRAFT, M.D.
Secretary

PAST OFFICERS OF THE ACADEMY

Date	President	First Vice-President	Treasurer	Secretary	Meeting Place
1896	Adolph Alt, M.D. St. Louis, Mo.	W. C. Pipino, M.D. Des Moines, Iowa	W. L. Dayton, M.D. Lincoln, Nebr.	Hal Foster, M.D. Kansas City, Mo.	Kansas City
1897	Adolph Alt, M.D. St. Louis, Mo.	W. C. Pipino, M.D. Des Moines, Iowa	W. L. Dayton, M.D. Lincoln, Nebr.	Hal Foster, M.D. Kansas City, Mo.	St. Louis
1898	B. E. Fryer, M.D. Kansas City, Mo.	J. E. Colburn, M.D. Chicago, Ill.	W. L. Dayton, M.D. Lincoln, Nebr.	Hal Foster, M.D. Kansas City, Mo.	Chicago
1899	J. E. Colburn, M.D. Chicago, Ill.	W. Scheppegrell, M.D. St. Louis, Mo.	W. L. Dayton, M.D. Lincoln, Nebr.	Thomas A. Woodruff, M.D. Chicago, Ill.	New Orleans
1900	W. Scheppegrell St. Louis, Mo.	M. A. Goldstein, M.D. St. Louis, Mo.	W. L. Dayton, M.D. Lincoln, Nebr.	Fayette C. Ewing, M.D. St. Louis, Mo.	St. Louis
1901	M. A. Goldstein, M.D. St. Louis, Mo.	H. W. Wurdemann, M.D. Milwaukee, Wis.	W. L. Dayton, M.D. Lincoln, Nebr.	W. L. Ballenger, M.D. Chicago, Ill.	Cincinnati
1902	C. R. Holmes, M.D. Cincinnati, Ohio	W. L. Dayton, M.D. Lincoln, Nebr.	Otto Stein, M.D. Chicago, Ill.	W. L. Ballenger, M.D. Chicago, Ill.	Chicago
1903	W. L. Ballenger, M.D. Chicago, Ill.	C. J. Stillson, M.D. Indianapolis, Ind.	Otto Stein, M.D. Chicago, Ill.	Derrick Vail, M.D. Cincinnati, Ohio	Indianapolis
1904	Edward Jackson, M.D. Denver, Colo.	Dudley Reynolds, M.D. Louisville, Ky.	Otto Stein, M.D. Chicago, Ill.	Derrick Vail, M.D. Cincinnati, Ohio	Denver
1905	Hanau Loeb, M.D. St. Louis, Mo.	Derrick Vail, M.D. Cincinnati, Ohio	Otto Stein, M.D. Chicago, Ill.	George F. Suker, M.D. Chicago, Ill.	Buffalo
1906	Casey Wood, M.D. Chicago, Ill.	J. A. Stucky, M.D. Lexington, Ky.	Otto Stein, M.D. Chicago, Ill.	George F. Suker, M.D. Chicago, Ill.	St. Clair
1907	J. A. Stucky, M.D. Lexington, Ky.	R. A. Reeve, M.D. Toronto, Canada	Otto Stein, M.D. Chicago, Ill.	George F. Suker, M.D. Chicago, Ill.	Louisville
1908	Derrick Vail, M.D. Cincinnati, Ohio	Joseph Beck, M.D. Chicago, Ill.	Otto Stein, M.D. Chicago, Ill.	George F. Suker, M.D. Chicago, Ill.	Cleveland
1909	Otto Stein, M.D. Chicago, Ill.	Percy Fridenberg, M.D. New York, N.Y.	S. H. Large, M.D. Cleveland, Ohio	George F. Suker, M.D. Chicago, Ill.	New York
1910	Wendell Reber, M.D. Philadelphia, Pa.	L. B. Lockhard, M.D. Denver, Colo.	S. H. Large, M.D. Cleveland, Ohio	George F. Suker, M.D. Chicago, Ill.	Cincinnati
1911	J. J. Kyle, M.D. Indianapolis, Ind.	F. Parke Lewis, M.D. Buffalo, N. Y.	S. H. Large, M.D. Cleveland, Ohio	George F. Suker, M.D. Chicago, Ill.	Indianapolis
1912	George F. Suker, M.D. Chicago, Ill.	J. W. Murphy, M.D. Cincinnati, Ohio	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Niagara Falls, Ont.
1913	J. W. Murphy, M.D. Cincinnati, Ohio	J. O. McReynolds, M.D. Dallas, Texas	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Chattanooga
1914	J. M. Ray, M.D. Louisville, Ky.	J. M. Ingersoll, M.D. Cleveland, Ohio	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Boston
1915	Joseph Beck, M.D. Chicago, Ill.	Robert S. Lamb, M.D. Washington, D.C.	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Chicago
1916	J. E. Brown, M.D. Columbus, Ohio	T. E. Carmody, M.D. Denver, Colo.	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Memphis
1917	W. L. Dayton, M.D. Lincoln, Nebr.	Edward Stieren, M.D. Pittsburgh, Pa.	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Pittsburgh
1918	Allen Greenwood, M.D. Boston, Mass.	Virginius Dabney, M.D. Washington, D. C.	S. H. Large, M.D. Cleveland, Ohio	L. M. Francis, M.D. Buffalo, N. Y.	Denver

PAST OFFICERS OF THE ACADEMY

Date	President	First Vice-President	Comptroller	Ex. Sec'y-Treas.	Meeting Place
1919	J. M. Ingersoll, M.D. Cleveland, Ohio	Melville M. Black, M.D. Denver, Colo.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Cleveland
1920	L. M. Francis, M.D. Buffalo, N. Y.	Hal Foster, M.D. Kansas City, Mo.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Kansas City
1921	Emil Mayer, M.D. New York, N. Y.	John R. Newcomb, M.D. Indianapolis, Ind.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Philadelphia
1922	Walter R. Parker, M.D. Detroit, Mich.	Ross H. Skillern, M.D. Philadelphia, Pa.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Minneapolis and St. Paul
1923	T. E. Carmody, M.D. Denver, Colo.	Hunter H. McGuire, M.D. Winchester, Va.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Washington, D. C.
1924	W. B. Lancaster, M.D. Boston, Mass.	William P. Wherry, M.D. Omaha, Nebr.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Montreal
1925	Horace Newhart, M.D. Minneapolis, Minn.	John M. Wheeler, M.D. New York, N. Y.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Chicago
1926	E. C. Ellett, M.D. Memphis, Tenn.	Austin A. Hayden, M.D. Chicago, Ill.	S. H. Large, M.D. Cleveland, Ohio	Luther C. Peter, M.D. Philadelphia, Pa.	Colorado Springs
1927	Ross Hall Skillern, M.D. Philadelphia, Pa.	H. W. Woodruff, M.D. Joliet, Ill.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Detroit
1928	Luther C. Peter, M.D. Philadelphia, Pa.	Frank L. Dennis, M.D. Colorado Springs, Colo.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	St. Louis
1929	Harris P. Mosher, M.D. Boston, Mass.	Frank E. Burch, M.D. St. Paul, Minn.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Atlantic City
1930	William H. Wilder, M.D. Chicago, Ill.	Frank R. Spencer, M.D. Boulder, Colo.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Chicago
1931	John F. Barnhill, M.D. Miami Beach, Fla.	Meyer Wiener, M.D. Coronado Calif.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	French Lick, Ind.
1932	S. Hanford McKee, M.D. Montreal, Canada	Gordon B. New, M.D. Rochester, Minn.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Montreal
1933	Burt R. Shurly, M.D. Detroit, Mich.	Nelson Miles Black, M.D. Miami, Fla.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Boston
1934	John M. Wheeler New York, N. Y.	LeRoy A. Schall, M.D. Boston, Mass.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Chicago
1935	Wells P. Eagleton, M.D. Newark, N. J.	W. Thornwall Davis, M.D. Washington, D. C.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Cincinnati
1936	Frank E. Burch, M.D. St. Paul, Minn.	Gordon F. Harkness, M.D. Davenport, Iowa	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	New York
1937	Lee W. Dean, M.D. St. Louis, Mo.	Bernard Samuels, M.D. New York, N. Y.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Chicago
1938	Harry S. Gradle, M.D. Chicago, Ill.	E. M. Seydell, M.D. Wichita, Kan.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Washington, D. C.
1939	George M. Coates, M.D. Philadelphia, Pa.	William W. Pearson, M.D. Des Moines, Iowa	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Chicago
1940	Frank E. Brawley, M.D. Chicago, Ill.	Arthur W. Proetz, M.D. St. Louis, Mo.	S. H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Cleveland
1941	Frank R. Spencer, M.D. Boulder, Colo.	Everett L. Goar, M.D. Houston, Texas	Secord H. Large, M.D. Cleveland, Ohio	William P. Wherry, M.D. Omaha, Nebr.	Chicago
1942	Ralph I. Lloyd, M.D. Brooklyn, New York	Walter H. Theobald, M.D. Chicago, Ill.	S. H. Large, M.D. Cleveland, Ohio	William L. Benedict, M.D. Rochester, Minn.	Chicago

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Alphabetical List of Members

Names and addresses corrected to March 15, 1943

Present membership role: Honorary, 20; Life, 252; Senior, 192; Junior, 2,903; Honorary Life Technical Consultant, 1; Total 3,368.

Number immediately following name indicates year in which the member was elected. Kindly check list and report errors to the Executive Secretary, William L. Benedict, M.D., 100 First Avenue Building, Rochester, Minnesota.

N. B.—By action of the Council at the 1934 meeting:

Fellows who have paid dues continuously for thirty (30) years will automatically become Life Members (designated by ⓛ), with dues remitted.

Fellows who have paid dues continuously for twenty-five (25) years will be termed Senior Members (designated by ⓢ), and their dues will be one-half the usual amount; at the present time, five dollars ($5.00) annually.

All other Fellows (except Honorary), (designated by ⓙ), will be classified as Junior Members. No designation carried.

N. B.—Present addresses of many members in active duty of the Armed Forces are not known and had to be thus designated in the following roster.

HONORARY LIFE TECHNICAL CONSULTANT

Max Poser.....635 St. Paul, Rochester, N. Y

A

Abbott, Edward A., '41, Lt., Camp Peary, Williamsburg, Va., **ALR**

Abbott, Albert Judson, '39, Capt. (M.C.), Valley Forge Gen. Hosp., Phoenixville, Pa., **OP**

Abell, Harry Davis, '42...333 Broadway, Paducah, Ky., **OP**

Abraham, Samuel Victor, '39.....1930 Wilshire blvd., Los Angeles, Cal., **OP**

Abrahamson, Ira A., '27, Lt. Col. (M.C.), A.P.O. 1142, San Francisco, Calif., **OP**

Ackerman, Walter Gabe, '39.....122 S. Michigan av., Chicago, Ill., **OP**

ⓛ**Adams, A. L.**, '06..Professional bldg., Jacksonville, Ill., **OALR**

Adams, Charles J., '28.....Armstrong-Landon bldg., Kokomo, Ind., **OALR**

Adams, D. S., '20.....Hume-Mansur bldg., Indianapolis, Ind., **OALR**

Adams, Eldridge S., '20.....Col. (M.C.), Station Hosp., Bowman Field, Ky., **ALR**

Adams, Frank M., '24..122 Waterman, Providence, R. I., **ALR**

Adams, Frederick W., '20.....1114 Boylston av., Seattle, Wash., **OALR**

Adams, John Goldthwaite, '38..24 Winter, Salem, Mass., **ALR**

Adamski, Michael Stanley, '40....8 Allen, Bath, N. Y., **OP**

Adelman, Benjamin B., '36.....190 Clinton av., Newark, N. J., **OP**

Adelstein, Fred, '41.....Rose bldg., Cleveland, Ohio, **ALR**

Adkins, George E., '29.....Jackson Infirmary, Jackson, Miss., **ALR**

Adler, Francis Heed, '29..313 S. 17th, Philadelphia, Pa., **OP**

Aebli, Rudolf, '31.....30 E. 40th, New York, N. Y., **OP**

Agan, William B., '30.....1 Nevins, Brooklyn, N. Y., **OP**

Agnew, Hobart McVicker, '38.....17 Plymouth, Montclair, N. J., **OP**

Albaugh, Clarence Henry, '41.....727 W. Seventh (Suite 1131), Los Angeles, Calif., **OP**

Albers, Edward C., '36.....104 W. Clark, Champaign, Ill., **OP**

Albright, George C., '31..104 S. Clinton, Iowa City, Ia., **ALR**

Albright, P. M., '22, Capt. (M.C.) U.S.N., U. S. Naval Hospital, Marine Barracks, Quantico, Va., **OALR**

Alden, Arthur M., '22.....Frisco bldg., St. Louis, Mo., **ALR**

ⓛ**Alderdyce, Wm. W.**, '09.....513 Madison av., Toledo, Ohio, **OALR**

Alexander, Charles S., '36.....Maj. (M.C.), Medical Arts bldg., Houston, Texas, **OP**

Alexander, Fred William, '42.....Capt. (M.C.), Station Hospital, Fort Dix, N. J., **ALR**

Alexander, Lucian W., '34.....Maison Blanche bldg., New Orleans, La., **ALR**

Alexander, Margaret H., '37.....720 Talbot, Taylor, Texas, **ALR**

Alexander, Samuel A., '33.....1830 Eye St. N.W., Washington, D. C., **ALR**

Alexander, S. L., '33..Medical Arts bldg., Toronto, Can., **ALR**

Alfaro, V. R., '31.....Capt. (M.C.), Station Hospital, Atlantic City, N. J., **ALR**

Alger, L. James, '36.....Box 328, Grand Forks, N. D., **ALR**

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Allen, Cary D., '36, Comdr. (M.C.), U. S. Naval Prison, Navy Yard, Portsmouth, N. H., **ALR**

Allen, Charles E., '20.....Waldheim bldg., Kansas City, Mo., **OALR**

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ⓛ**Allen, J. H.**.....2081 Garfias Drive, Pasadena, Calif., **ALR**

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Allen, Orris T., '28.....Rose Dispensary bldg., Terre Haute, Ind., **OALR**

Allen, Thomas Dyer, '28.....122 S. Michigan av., Chicago, Ill., **OP**

Allis, Paul M., '36.....6 N. Brown, Lewistown, Pa., **ALR**
Allman, Charles Hugh, '41, Comdr. (M.C.), U.S.S. Richmond, c/o Postmaster, San Francisco, Calif., **ALR**
Almour, Ralph, '25.....In Service (address unknown) **ALR**
Altringer, A. N., '20.....Professional bldg., Kansas City, Mo., **ALR**
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Alvis, Raymond W., '40.....Ohio bldg., Sidney, Ohio, **OALR**
Amberg, Emil, '06.1244 W. Boston blvd., Detroit, Mich., **ALR**
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Amsel, Maxwell Ritter, '38.....145 W. 55th St., New York, N. Y., **ALR**
Anderson, Edwin Rudolph, '38.220 Liberty, Warren, Pa., **OALR**
Anderson, George Henry, '40...Capt. (M.C.), Station Hospital, Fort Knox, Ky., **ALR**
Anderson, Orrin Emanuel, '41, Capt. (M.C.), Gen. Disp. N.S.A., 39 White Hall st., New York, N. Y., **ALR**
Anderson, Ross Epting, '41.....Standard Life bldg., Jackson, Miss., **ALR**
Anderson, Ruskin Gregory, '41, Capt. (M.C.), Kennedy Gen. Hosp., Memphis, Tenn., **OP**
Anderson, W. B., '13.....309 W. Anderson, Brownwood, Texas, **OALR**
Anderson, Wm. Banks, '35.....Duke Hospital, Durham, N. C., **OP**
Andrews, A. H., '98....25 E. Washington, Chicago, Ill., **OALR**
Andrews, Albert H., Jr., '36, Lt. (M.C.), Med. Research Sec., Bureau of Aeronautics, Washington, D. C., **ALR**
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Andrews, M. P., '15.....826 S. 8th, Manitowoc, Wis., **OALR**
Anneberg, A. Reas, '39, Capt. (M.C.), 37 Station Hosp., Camp Barkeley, Texas, **ALR**
Annon, W. T., '23.....235 S. 15th, Philadelphia, Pa., **ALR**
Anthony, Daniel H., '29.....Exchange bldg., Memphis, Tenn., **OP**
Appelbaum, Alfred, '36.....Maj. (M.C.), Station Hospital, Camp Kilmer, N. J., **OP**
Apple, Carl, '38.....55 E. Washington, Chicago, Ill., **OP**
Appleman, L. F., '13.....308 S. 16th, Philadelphia, Pa., **OP**
Arbuckle, David S., '31.....Akron S. & L. bldg., Akron, Ohio, **OALR**
Arbuckle, Millard F., '15.....Col. (M.C.), Sta. Hosp., Fort Leonard Wood, Mo., **ALR**
Armentrout, Lewis Hamilton, Jr., '42.....Grace Hospital, Welch, W. Va., **ALR**
Armstrong, E. Leslie, '32.....Medical Arts bldg., Duluth, Minn., **ALR**
Armstrong, R. M., '28.....1st Nat'l Bank bldg., Lexington, Ky., **OALR**
Arnold, Francis J., '12.....182 Pearl, Burlington, Vt., **OALR**
Arnold, Morton, '38.....781 Main, Willimantic, Conn., **ALR**
Arnold, Ralph A., '42, Capt. (M.C.), ...65th General Hospital, Fort Bragg, N. C., **ALR**
Aronson, Sidney S., '29....Capt. (M.C.), Station Hospital, Smyrna, Tenn., **ALR**
Arrell, W., '21...317 Main St. E., Hamilton, Ont., Can.,**OALR**
Asbill, David St. Pierre, '33, Capt. (M.C.), Walter Reed Hospital, Washington, D. C., **OP**
Asbury, Mary K., '25.3549 Holly Lane, Cincinnati, Ohio, **OP**
Ashley, Byron J., '37.....901 Kansas av., Topeka, Kan., **ALR**
Ashley, Rea Ernest, '38.....U. S. Naval Hospital, Corona, Calif., **ALR**
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Atkins, John Leon, '39.....345 Bloor St. West, Toronto, Ont., Can., **ALR**
Atkinson, D. T., '18.....Medical Arts bldg., San Antonio, Texas, **OALR**
Atkinson, John H., '36. 168 Sterling, Watertown, N. Y., **ALR**
Atkinson, Miles, '38.....123 E. 61st, New York, N. Y., **ALR**
Atkinson, Thomas E., '29.....Coronado bldg., Greeley, Colo., **ALR**
Atkinson, Walter S., '25.129 Clinton, Watertown, N. Y., **OP**
Atwood, Edward A., '32...360 Park av., Paterson, N. J., **ALR**
Auerbach, Julius, '20...897 Park av., New York, N. Y., **ALR**
Austen, Willard Emerson, '40, Lt. Comdr. (M.C.), 115 East 61st, New York, N. Y., **ALR**
Austin, Marvin Fletcher, '42, Maj. (M.C.), 231 Main st., Hempstead, N. Y., **ALR**
Avery, Elroy J., '34..11 N. Goodman, Rochester, N. Y., **ALR**
Awtrey, Hugh Hanna, '39, Lt. Comdr. (M.C.), U. S. Naval Hosp., Treasure Island, San Francisco, Calif., **ALR**
Aynesworth, H. T., '12.Medical Arts bldg., Waco, Texas, **OALR**

B

Babbitt, James A., '24...1912 Spruce, Philadelphia, Pa., **ALR**
Bach, Edwin Clarence, '40.....324 E. Wisconsin av., Milwaukee, Wis., **ALR**
Bacon, Lewis Heisler, '42, Lt. Comdr. (M.C.), Naval Medical Center, Bethesda, Md., **OP**
Baer, Alvin Julius, '42, Maj. (M.C.), Station Hospital, Jefferson Barracks, Mo., **OP**
Baeseman, R. Winfield, '29.....501 Grand av., Asbury Park, N. J., **OALR**
Bagley, Cecil Hopkins, '41.2 East Read, Baltimore, Md., **OP**
Bahn, Charles A., '22.....Pere Marquette bldg., New Orleans, La., **OP**
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Bailey, Paul, '37, Maj. (M.C.), McCaw General Hospital, Walla Walla, Wash., **ALR**
Baird, Charles G., '20...Col. (M.C.), 130 Station, Camp Barkeley, Texas, **OALR**
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Baker, Melvin C., '40...Heyburn bldg., Louisville, Ky., **OP**
Bakst, Max, '38....186 New York av., Brooklyn, N. Y., **ALR**
Balding, Laurence Grant, '41.....17 Field Hospital, Camp Bowie, Texas, **OP**
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Baldwin, Stephen Glidden, '42.....Adams bldg., Danville, Ill., **ALR**
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Ball, Simon, '36.....1920 Pine, Philadelphia, Pa., **ALR**
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Ballon, D. H., '24.....1471 Crescent, Montreal, Can., **ALR**
Bane, W. M., '20.....1612 Tremont pl., Denver, Colo., **OP**

Bankes, Claude Webster, '38. 212 N. 6th, Reading, Pa., **OP**
Barber, F., '13.....75 S. Fitzhugh, Rochester, N. Y., **OP**
Barbour, Fleming Arnold, '41.....
.....1816 Highland pl., Berkeley, Calif., **OP**
Bardenheier, F. G. A., '14...Frisco bldg., St. Louis, Mo., **ALR**
Barkan, Hans , '18.....
.....1940 Broadway st., San Francisco, Calif., **OP**
Barkan, Otto, '29.....490 Post, San Francisco, Calif., **OP**
Barkhoen, Charles W., '33, Maj. (M.C.),
Percy Jones Gen. Hosp., Battle Creek, Mich., **ALR**
Barlow, Aaron, '36.....1307 Spruce, Philadelphia, Pa., **OP**
Barlow, Roy A., '20..Box 333, Charles Town, West Va., **ALR**
Barnes, Lancelot, '35.....170 St. George, Toronto, Can., **OP**
Barnes, Mark Hopkins, '42.....
.....324 E. 41st st., New York, N. Y., **OALR**
Barnett, Irving F., '33.....
.....30 N. Michigan blvd., Chicago, Ill., **OALR**
Barnett, Saul E., '29.....25 Parsons, Detroit, Mich., **ALR**
Baron, S. H., '35, Lt. Comdr. (M.C.), U.S.N.R.,
Navy 608, Fleet Post Office, San Francisco, Calif., **ALR**
Barone, Michael Horace, '42, Capt. (M.C.),
27th Field Hospital, Camp Campbell, Ky., **ALR**
Barr, A. S., '32.1st Nat'l Bank bldg., Ann Arbor, Mich., **OP**
Barr, William T., '38.....
Capt. (M.C.), Air Base Hospital, Marfa, Texas, **ALR**
Barry, William B., '42.....
Univ. of Kansas Hospitals, Kansas City, Kans., **ALR**
Bartley, Donald A., '39.....
.....Hume-Mansur bldg., Indianapolis, Ind., **OP**
Barton, John F., '31.....
.....1346 Fourteenth av., Longview, Wash., **ALR**
Barton, William C., '32.....
.....104½ W. San Francisco, Santa Fe, N. M., **OALR**
Bass, A. L., '24.....Heyburn bldg., Louisville, Ky., **OALR**
Bassen, Edward J., '30.....
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Bassett, David I., '40.....
.....1075 Grand Concourse, New York, N. Y., **ALR**
Batson, Oscar V., '34.....133 S. 36, Philadelphia, Pa., **ALR**
Battles, N. H., '37.....
Capt. (M.C.), 311 N. Placer av., Idaho Falls, Idaho, **OALR**
Baum, H. L., '17.....Republic bldg., Denver, Colo., **ALR**
Baum, Wm. W., '30, Maj. (M.C.),
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Bausch, Carl Philipp, '38.....36 Pearl, Hartford, Conn., **OP**
Beach, S. Judd, '14.....704 Congress, Portland, Maine, **OP**
Beal, Homer A., '26, In Service, Professional bldg.,
Kansas City, Mo., **ALR**
Beale, Allen William, '40.....
.....A.P.O. 886, c/o Postmaster, New York, N. Y., **ALR**
Beals, C. Wearne, '30.....41 N. Brady, DuBois, Pa., **ALR**
Beam, A. Duane, '39.....
Lt. (M.C.), U. S. Naval Hospital, Philadelphia, Pa., **OP**
Beattie, Robert, '06.....
.....David Whitney bldg., Detroit, Mich., **OALR**
Beattie, William H., '21.....258 Genesee, Utica, N. Y., **OALR**
Beatty, H. G., '16.....150 E. Broad, Columbus, Ohio, **ALR**
Beavis, James Olin, '36.....
.....900 Fidelity Medical bldg., Dayton, Ohio, **ALR**
Beck, A. L., '27....421 Huguenot, New Rochelle, N. Y., **OALR**
Beck, Frederick White, '40.....
Capt. (M.C.), Deshon General Hospital, Butler, Pa., **ALR**
Beck, Robert J., '35....105 Main, Saranac Lake, N. Y., **OP**
Becker, Oscar J., '41.310 S. Michigan av., Chicago, Ill., **ALR**
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.....1050 Fifth av., Huntington, W. Va., **ALR**
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Beem, Ione F., '16.....
.....30 N. Michigan av., Sec. 2, Chicago, Ill., **OALR**
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.....2730 E. Jefferson av., Detroit, Mich., **OP**
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.....9 Prospect Park West, Brooklyn, N. Y., **OP**
Beideman, J. E., '29.....
.....Montgomery Trust Arcade, Norristown, Pa., **OP**
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.....760 Eastern Parkway, Brooklyn, N. Y., **ALR**
Beisbarth, Carl, '31.....
.....3720 Washington blvd., St. Louis, Mo., **OP**
Bell, John Gordon, '40.....
.....1835 Eye Street, N. W., Washington, D. C., **ALR**
Bell, Julius W., '36.....200 E. 16th, New York, N. Y., **ALR**
Bell, Will Otto, '28. Medical-Dental bldg., Seattle, Wash., **OP**
Bellows, John G., '35.720 N. Michigan av., Chicago, Ill., **OP**
Bender, H. A., '30.....Black bldg., Waterloo, Iowa, **ALR**
Bender, M. S., '30.....88-05 150th st., Jamaica, N. Y., **ALR**
Also: 121 E. 60th, New York, N. Y.,
Benedict, William L., '19. Mayo Clinic, Rochester, Minn., **OP**
Benkwith, Karl Burton, '42, Lt. (M.C.), U. S. Naval
Hospital, Marine Barracks, Quantico, Va., **OP**
Benson, Clifton Emanuel, '40.....
Lt. Comdr. (M.C.), 1021 Redgate av., Norfolk, Va., **ALR**
Bentley, Frederick, '37.....Cobb bldg., Seattle, Wash., **OP**
Bentley, Neil I., '22.....
.....David Whitney bldg., Detroit, Mich., **OALR**
Benz, Alvin Hermann, '41, Capt. (M.C.), Sta. Hosp.,
N.O.P.E., Jefferson Branch, New Orleans, La., **OP**
Berens, Conrad, '22.....35 E. 70th, New York, N. Y., **OP**
Berger, Isidore, '40.....
.....888 Grand Concourse, Bronx, New York, N. Y., **ALR**
Berger, Louis, '40.....30 N. Michigan av., Chicago, Ill., **OP**
Berger, Murray, '38.....
.....300 Central Park West, New York, N. Y., **ALR**
Bergmann, M. B., '32.....
.....959 Bushwick av., Brooklyn, N. Y., **OP**
Berke, Raynold N., '34....430 Union, Hackensack, N. J., **OP**
Berliner, Milton L., '31...57 W. 57th, New York, N. Y., **OP**
Berman, Philip G., '35.....174 Central, Lowell, Mass., **OP**
Bernatz, C. F., '17.....Park bldg., Pittsburgh, Pa., **OP**
Berner, George E., '37...5430 Greene, Philadelphia, Pa., **OP**
Bernheimer, L. Benno, '30.....
.....104 S. Michigan av., Chicago, Ill., **ALR**
Berns, Charles, '35, Maj. (M.C.), 34th Evacuation Hosp.,
Camp Barkeley, Texas, **ALR**
Berry, D. F., '11. Bankers Trust bldg., Indianapolis, Ind., **ALR**
Berry, Gordon, '30.....36 Pleasant, Worcester, Mass., **ALR**
Bertolet, John A., '37....1530 Locust, Philadelphia, Pa., **ALR**
Bertrand, George Joseph, '42.....
.....73 Main st., Montpelier, Vt., **OP**
Best, L. K., '35.....151 Rock, Fall River, Mass., **OP**
Beyer, A. G., '25.....Carew Tower, Cincinnati, Ohio, **OALR**
Beyer, Louis J., '18...449 Delaware av., Buffalo, N. Y., **ALR**
Beyer, Theodore E., '31.227 Sixteenth st., Denver, Colo., **ALR**
Biehn, J. Telford, '37, Capt. (R.C.A.M.C.),
Military Hospital, Camp Borden, Ont., **OALR**
Bilchick, Edwin B., '35.....
.....Maj. (M.C.), 216 W. 89th, New York, N. Y., **ALR**
Billings, Gilbert M., '39.....
.....110 S. Sterling, Morganton, N. C., **ALR**
Billings, Robert J., '35....500 Penn av., Pittsburgh, Pa., **OP**

Billingsley, John A., '38
Huron bldg., Kansas City, Kan., **OP**

Binger, H. E., '17 Lowry Medical bldg., St. Paul, Minn., **OALR**

Binkley, Roy S., '17 Fidelity bldg., Dayton, Ohio, **OALR**

Birch, John Randolph, '39
Maccabees bldg., Detroit, Mich., **ALR**

Birdsall, Clarence H., '37 30 Summer, Haverhill, Mass., **ALR**

Birge, Henry, '39
Capt. (M.C.), A.F.A.T.S., Stockton Field, Calif., **OP**

Birsner, Louis J., '26
6045 Waterman av., St. Louis, Mo., **ALR**

Bishop, Frederick J., '36
Medical Arts bldg., Scranton, Pa., **ALR**

Bishop, Vernon L., '33 277 Alexander, Rochester, N. Y., **ALR**

Blauuw, E. E., '05 190 Ashland av., Buffalo, N. Y., **OP**

Black, Nelson Miles, '03 Huntington bldg., Miami, Fla., **OP**

Black, Paul, '27 Stuart bldg., Lincoln, Nebr., **OALR**

Black, W. Byron, '31
Professional bldg., Kansas City, Mo., **ALR**

Black, W. D., 1900- Metropolitan bldg., St. Louis, Mo., **ALR**

Blackburn, W. J., '19 Reibold bldg., Dayton, Ohio, **OALR**

Blackmar, Francis B., '31
Maj. (M.C.), Schick Gen. Hosp., Clinton, Iowa, **ALR**

Blair, M. W., '28
N. E. cor. 20th & Chestnut, Philadelphia, Pa., **OP**

Blair, W. W., '17 Woodland road, (6), Pittsburgh, Pa., **OP**

Blaisdell, Irl Hazard, '41
Medical Arts bldg., Syracuse, N. Y., **ALR**

Blakesley, T. S. Professional bldg., Kansas City, Mo., **OALR**

Blankfeld, Nathan, '36
429 Lincoln pl., Sta. B, Brooklyn, N. Y., **ALR**

Blankstein, Samuel S., '42
606 Wisconsin av., Milwaukee, Wis., **OP**

Blasdel, Tyler David, '40
110½ S. 18th st., Parsons, Kan., **ALR**

Blassingame, C. D., '23
899 Madison av., Memphis, Tenn., **ALR**

Bledsoe, R. W., '07
First Nat'l Bank bldg., Covington, Ky., **OALR**

Blickenstaff, A. J., '22 Alliance Life bldg., Peoria, Ill., **OALR**

Blomberg, Thorsten E., '33
501 Seventh st., Rockford, Ill., **ALR**

Blonder, Edwin J., '34 185 N. Wabash av., Chicago, Ill., **ALR**

Bloom, C. Hugh, '32 65 N. 3rd, Easton, Pa., **ALR**

Blue, J. B., '16 62 N. Main, Memphis, Tenn., **OALR**

Blum, Henry N., '14
American Bank bldg., New Orleans, La., **OP**

Blumenfeld, Louis, '39
221 Brooklyn av., Brooklyn, N. Y., **ALR**

Boaz, Emmett Daniel, '38
3710 Fifth av., Pittsburgh, Pa., **ALR**

Bochner, Maxwell K., '33
329 Bloor st. W., Toronto, Can., **OP**

Bockoven, Sterling, '38
1752 Mass. av. N.W., Washington, D. C., **OP**

Boder, Worthey Carl, '42
Capt. (M.C.), Keesler Field, Biloxi, Miss., **ALR**

Boeckmann, Egil, '18
Lowry Med. Arts bldg., St. Paul, Minn., **OALR**

Boehs, Charles J., '37
Medical Arts bldg., San Antonio, Texas, **ALR**

Boemer, L. C., '30, Maj. (M.C.),
In Service, (address unknown), **ALR**

Bogart, Donald Weeks, '40
20 East 53rd st., New York, N. Y., **OP**

Boggs, Frank Clinton, '38 Mills bldg., Topeka, Kan., **OP**

Boice, Ralph E., '35 316 Michigan, Toledo, Ohio, **OP**

Boles, Lawrence R., '36
Medical Arts bldg., Minneapolis, Minn., **ALR**

Boiler, W. F., '12
66 Rolla Gardens, Rolla, Mo., **OALR**

Boland, William Tilden, '41 315 Lake, Elmira, N. Y., **OP**

Boles, William McDonald, '41
Capt. (M.C.), 24 Gen. Hosp., Fort Benning, Ga., **OP**

Bolman, Harold Robert, '38
6½ S. Main, Monroeville, Ohio, **OP**

Bolotow, Nathan A., '29
126 Waterman, Providence, R. I., **ALR**

Bonaccolto, Girolamo, '37
654 Madison av., New York, N. Y., **OP**

Bond, Floyd M., '41 625 Broadway, San Diego, Calif., **OP**

Bone, Herman D., '20 19 Pleasant, Gardner, Mass., **OALR**

Bonham, William Lawrence, '39
Medical Arts bldg., Oklahoma City, Okla., **ALR**

Bonner, Wm. F., '26
Veterans Administration Facility, Batavia, N. Y., **OALR**

Bonner, William Richard, '42
3 W. White st., Summit Hill, Pa., **OP**

Bordley, James, Jr., '18
4 Charleote pl., Baltimore, Md., **OALR**

Borgeson, E. J., '30
Medical Arts bldg., Minneapolis, Minn., **OP**

Borley, William E., '39 2351 Clay, San Francisco, Calif., **OP**

Bornstein, Max, '42, Maj. (M.C.), Post Hospital,
1570 S.U., Camp Breckinridge, Ky., **ALR**

Boss, Eugene Geo., '33 1537 Main, Springfield, Mass., **OALR**

Bostwick, D. S., '34 106 Linwood av., Ardmore, Pa., **ALR**

Boswell, Robert Echo, '41 Fidelity bldg., Dayton, Ohio, **ALR**

Bothman, Louis, '30 310 S. Michigan av., Chicago, Ill., **OP**

Boucher, R. B., '19
Medical-Dental bldg., Vancouver, B. C., Can., **OALR**

Bourbon, Oliver P., '20 520 W. 7th, Los Angeles, Calif., **OALR**

Bourne, Malcolm G., '37 110 W. 3rd, Jamestown, N. Y., **OP**

Bouvy, Harry M., '20
921 S. W. Washington, Portland, Ore., **OALR**

Bouvy, L. B., '27 Foley bldg., La Grande, Ore., **OALR**

Bowers, Wesley C., '36 17 E. 61st, New York, N. Y., **ALR**

Boyce, G. H., '12
1st Nat'l Bank bldg., Iron Mountain, Mich., **OALR**

Boyce, Wm. A., '31 Roosevelt bldg., Los Angeles, Calif., **OP**

Boyd, Charles Wilson, '40
111 West Adams st., Jacksonville, Fla., **OP**

Boyd, Greydon Gill, '39
Lt. Comdr. (M.C.), Marine Air Sta., El Toro, Calif., **ALR**

Boyden, Guy Lee, '39 1735 N. Wheeler, Portland, Ore., **ALR**

Boyes, T. L., '32 654 Madison av., New York, N. Y., **OP**

Bozer, Herrmann E., '23
468 Delaware av., Buffalo, N. Y., **ALR**

Bradford, George Edwin, '41
Nissen bldg., Winston-Salem, N. C., **ALR**

Bradish, Robert Francis, Maj., (M.C.), New Orleans Port
of Embarkation, Poland and Dauphin sts., New
Orleans, La., **OP**

Bradley, Jeter C., '34
900 17th st. N. W., Washington, D. C., **ALR**

Bradley, John W., '37
Maj. (M.C.), Sta. Hosp., Camp Young, Indio, Calif., **ALR**

Braley, Alson E., '39 630 W. 168, New York, N. Y., **OP**

Brandenburg, Kenneth C., '37
110 Pine av., Long Beach, Calif., **OALR**

Brandenburg, Nora B., '36
720 N. Michigan av., Chicago, Ill., **ALR**

Brandfield, Ira Irving, '40
Capt. (M.C.), Station Hospital, Camp Stewart, Ga., **ALR**

Brandwan, Samuel R., '37
1851 E. 6th st., Cleveland, Ohio, **OP**
Branigan, C. Hugh, '33...114 E. 54th, New York, N. Y., **OP**
Brannin, Daniel, '36...Medical Arts bldg., Dallas, Texas, **OP**
Braswell, James C., '22..Medical Arts bldg., Tulsa, Okla., **OALR**
Braunlin, Wm. H., '20
Marion Nat'l Bank bldg., Marion, Ind., **OALR**
◐Brawley, F. E., '07....30 N. Michigan av., Chicago, Ill., **OP**
Brawner, Leon E., '29..Medical Arts bldg., Atlanta, Ga., **ALR**
Bredlau, Ernest A., '29....119 LeMoyne, Oak Park, Ill., **ALR**
Breeding, Earl G., '32
1801 Eye st. N. W., Washington, D. C., **ALR**
Brehmer, Harrison L., '20
First Nat'l Bank bldg., Albuquerque, N. M., **OALR**
Breitstein, M. Lewis, '37
1213 Eutaw pl., Baltimore, Md., **ALR**
Bressler, J. L., '34..Medical Arts bldg., Houston, Texas, **OP**
Breuning, Paul H., '32...133 E. 58th, New York, N. Y., **ALR**
Brewer, John D., '20
Baird Brewer Gen. Hosp., Dyersburg, Tenn., **OALR**
Brewster, H. F., '32
Union Indemnity bldg., New Orleans, La., **ALR**
Briant, Thomas E., '29..28 E. Main, Welland, Ont., Can., **ALR**
Bribach, Eugene J., '19
Professional bldg., Atchison, Kan., **OALR**
Bricker, Sacks, '31..1101 W. Wyoming, Philadelphia, Pa., **ALR**
Brickley, Daniel W., '21
Uhler Phillips bldg., Marion, Ohio, **OALR**
Brickley, Daniel Webster, Jr., '38
Uhler-Phillips bldg., Marion, Ohio, **ALR**
Bridgett, Frank Aloysius, '42
3330 Chestnut st., Philadelphia, Pa., **ALR**
Briggs, Henry Harrison, Jr., '41
Maj. (M.C.), 3064 Puritan st., Lincoln, Nebr., **OP**
Brighton, George R., '36..580 Park av., New York, N. Y., **ALR**
Briglia, Frank J., '36...819 Wharton, Philadelphia, Pa., **ALR**
Brindley, Benjamin, '40
Lt. Comdr., U. S. Naval Hosp., Balboa, Canal Zone, **OP**
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329 Professional bldg., Phoenix, Ariz., **ALR**
Bristow, Walter J., '35..1517 Hampton, Columbia, S. C., **OALR**
Broadwell, Stuart, Jr., '37..103½ N. 5th, Springfield, Ill., **ALR**
Brobeck, von Haller, '32
Ferguson bldg., Colorado Springs, Colo., **OP**
◐Brobst, C. H., '06.....107 High st., Peoria, Ill., **OALR**
◐Brodrick, Frank W., '11.....302 First av., Sterling, Ill., **OALR**
Brook, Jeffrey J., '34
231 W. Wisconsin av., Milwaukee, Wis., **ALR**
◑Brooks, E. H., '15.....Zuelke bldg., Appleton, Wis., **OALR**
Brooks, Earl B., '21....128 N. 13th st., Lincoln, Nebr., **OALR**
Brooks, Morris J., '34..455 Ocean av., Brooklyn, N. Y., **ALR**
Brooks, Richmond C. A., '39
1150 Connecticut av., Washington, D. C., **ALR**
Broones, Otto, '36.....302 W. 87th, New York, N. Y., **ALR**
Brown, Albert L., '26....Carew Tower, Cincinnati, Ohio, **OP**
Brown, Andrew George, '42
1010 Huntington bldg., Miami, Fla., **OP**
Brown, Audrey O., '27..5057 Woodward, Detroit, Mich., **OALR**
Brown, Charles Lafayette, '38
Union bldg., New Orleans, La., **ALR**
Brown, Charles Wm., '27
Bank of America bldg., San Diego, Calif., **ALR**
Brown, C. J., '34, Captain (M.C.) U.S.N.,
Naval Hospital, Philadelphia, Pa., **ALR**
◐Brown, Clayton M., '12..510 Delaware av., Buffalo, N. Y., **ALR**
Brown, Clyde O., '28
3720 Washington blvd., St. Louis, Mo., **ALR**
Brown, David Edward, '41
23 E. Ohio, Indianapolis, Ind., **ALR**
Brown, Earl Henderson, '39..130 S. Scott, Tucson, Ariz., **ALR**
◐Brown, J. M., '11.....1136 W. 6th, Los Angeles, Calif., **ALR**
Brown, John C., '20...Medical Arts bldg., St. Paul, Minn., **OALR**
◐Brown, John E., '01...370 East Town, Columbus, Ohio, **OALR**
Brown, Lester A., '39
478 Peachtree st. N. E., Atlanta, Ga., **ALR**
Brown, Morrow D., '20...Republic bldg., Denver, Colo., **ALR**
Brown, Mortimer G., '21
State Tower bldg., Syracuse, N. Y., **ALR**
◐Brown, W. Harold, '12
Birks bldg., Vancouver, B. C., Can., **OALR**
Brownell, Durwin Hall, '40
Medico-Dental bldg., San Diego, Calif., **ALR**
Brownell, Morton E., '28...105 N. Main, Wichita, Kan., **OP**
Browning, Andrew J., '35....Mayer bldg., Portland, Ore., **OP**
Browning, Birt Lee, '35...208 Grand, Newburgh, N. Y., **ALR**
Brownsberger, Sidney Burten, '38, Capt. (M.C.),
Letterman Gen. Hosp., San Francisco, Calif., **OP**
Bruce, G. M., '30
Lt. Comdr. (M.C.), 635 W. 165th, New York, N. Y., **OP**
◐Bruder, Joseph, '08.....57 W. 57th, New York, N. Y., **OALR**
Brumbaugh, John D., '34
2nd Nat'l Bank bldg., Akron, Ohio, **OALR**
Brumm, Seth A., '22
Medical Arts bldg., Philadelphia, Pa., **OALR**
Bruner, Abram B., '23...Guardian bldg., Cleveland, Ohio, **OP**
Bruner, Claude R., '41
Lt. Comdr., Fleet Postmaster, San Francisco, Calif., **OP**
◐Bruner, Wm. E., '05...Guardian bldg., Cleveland, Ohio, **OP**
Brunk, Clifford F., '39
7815 E. Jefferson av., Detroit, Mich., **ALR**
Brunner, George Harmon, '40
McCannel Clinic bldg., Minot, N. D., **OP**
◑Brunner, Hans, '35.....1853 Polk, Chicago, Ill., **ALR**
Bruno, Joseph M. L., '35..31 Jefferson, Brooklyn, N. Y., **OP**
Brusegard, James F., '33..414 Main, Red Wing, Minn., **OP**
◐Brust, H. O., '13....820 S. Crouse av., Syracuse, N. Y., **ALR**
Bryan, James L., '28
Bennie-Dillon bldg., Nashville, Tenn., **OALR**
Bryan, William T. K., '42
3720 Washington blvd., St. Louis, Mo., **ALR**
Bryant, Ben L., '31
2312 Miramar st., Los Angeles, Calif., **ALR**
Bryant, Frank L., '37..1600 W. Lake, Minneapolis, Minn., **ALR**
Buchanan, Norman D., '25
129 Murray, Peterborough, Ont., Can., **OALR**
Bucher, Walter M., '34..4187 Pearl road, Cleveland, Ohio, **ALR**
Buck, Clair A., '31
Maj. (M.C.), 191 Glen, Glens Falls, N. Y., **ALR**
Buckingham, Tracy W., '31
405 Broadway, Bismarck, N. D., **ALR**
Buckman, Lewis T., '32
83 S. Franklin, Wilkes-Barre, Pa., **OALR**
Buckman, Samuel Thompson, '40
70 S. Franklin, Wilkes-Barre, Pa., **ALR**
Buffington, Wiley R., '35
Hibernia Bank, New Orleans, La., **OP**
Buffum, Edward S., '36....40 W. North, Buffalo, N. Y., **ALR**
Bullard, Carleton Wm., '32..120 Genesee, Auburn, N. Y., **ALR**
Bullis, John Albert E., '39
1136 W. Sixth, Los Angeles, Calif., **OALR**
Bullwinkel, H. Griffin, '31
Comdr. (M.C.), Bronson Field, Pensacola, Fla., **ALR**
Bulson, Eugene L., '28..347 W. Berry, Ft. Wayne, Ind., **OALR**
Bunker, Paul G., '37..Newberry bldg., Aberdeen, S. D., **ALR**

Bunnell, Chester William, '35.....
...Lt. Comdr. (M.C.), 9115 193rd st., Hollis, N. Y., **ALR**
Buonaguro, Michael J., '31.....
.....589 Lorimer, Brooklyn, N. Y., **OP**
Burch, Edward P., '39, Maj. (M.C.), O'Reilly General
Hospital, Springfield, Mo., **OP**
Burch, Frank E., '07...754 Linwood pl., St. Paul, Minn., **OP**
Burchfield, George W., '36..309 Court, Maryville, Tenn., **ALR**
Burdick, Austin F., '25.....
.....P. O. Drawer 1378, Lansing, Mich., **OALR**
Burgess, John Louis, '40.....
.....Medical Arts bldg., Waco, Texas, **ALR**
Burgess, Taylor S., '39...Medical Arts bldg., Atlanta, Ga., **ALR**
Burgman, James Everette, '38.....
.....8 W. Main, Springfield, Ohio, **ALR,**
Burian, Hermann M., '38.....
.....The Eye Clinic, Hanover, N. H., **OP**
Burke, B. Russell, '35, Maj. (M.C.), 43rd General
Hospital, Camp Livingston, La., **ALR**
Burke, John R., '27.....207 Elm, Holyoke, Mass., **OALR**
Burke, T. A., '06.....Rose bldg., Cleveland, Ohio, **OALR**
Burman, Herman J., '35.....
.....1235 Grand Concourse, New York, N. Y., **ALR**
Burnham, Howard H., '37.....
.....Medical Arts bldg., Toronto, Can., **ALR**
Burns, L. J., '30.....1930 Chestnut, Philadelphia, Pa., **ALR**
Burns, S. S., '19..Missouri Theatre bldg., St. Louis, Mo., **ALR**
Burritt, N. W., '30...30 Beechwood road, Summit, N. J., **ALR**
Burton, Edwin W., '37.....
.....Y.M.C.A. bldg., Charlottesville, Va., **OP**
Bushman, L. B., '27.....
.....City Nat'l Bank bldg., Omaha, Nebr., **OALR**
Butler, Edmund John, '42.....
.....25 Garden st., Cambridge, Mass., **ALR**
Butler, Harry, '37.....
....Maj. (M.C.), A.F.T.T.S., Sioux Falls, So. Dak., **ALR**
Butler, Robert H., '20.....
.....112 W. Columbus av., Bellefontaine, Ohio, **OALR**
Butt, Miriam M., '08...2045 Chestnut, Philadelphia, Pa., **OP**
Buvinger, Charles W., '21.....
.....50 Washington, East Orange, N. J., **OALR**
Buzzard, Josiah F., '31.....
.....1110 Thirteenth av., Altoona, Pa., **OALR**
Byrne, Harry Vincent, '38...301 Essex, Lawrence, Mass., **OP**
Byrne, J. Arthur, '41.....16 Elm, Morristown, N. J., **ALR**
Byrnes, Harry F., '10...67 Chestnut, Springfield, Mass., **OALR**

C

Cabot, Clyde Marcus, '38.....
.....948 Medical Arts bldg., Minneapolis, Minn., **ALR**
Cady, D. W., '30...65 N. Madison av., Pasadena, Calif., **OP**
Cady, F. J., '32....402 S. Jefferson av., Saginaw, Mich., **OALR**
Cahill, James F., '37.....428 S. Salina, Syracuse, N. Y., **OP**
Calcote, Royal J., '36, Lt. Comdr. (M.C.), Naval Officer
Procurement, 703 Market st., San Francisco, Calif., **OP**
Caldwell, Robert, '12.....
.....New Donaghey bldg., Little Rock, Ark., **OALR**
Calhoun, F. Phinizy, '08.....
.....478 Peachtree st. N.E., Atlanta, Ga., **OP**
Callahan, Alston, '38.....
.....144 Ponce de Leon av. N.E., Atlanta, Ga., **OP**
Callfas, W. F., '19.....525 Spruce, San Diego, Calif., **ALR**
Calvert, Raymond R., '36.....
.....Maj. (M.C.), Selfridge Field, Mich., **OP**
Camp, W. E., '20.....
.....Medical Arts bldg., Minneapolis, Minn., **OALR**
Campbell, A. A., '30.....86 Bloor st. W., Toronto, Can., **ALR**
Campbell, C. H., '18.....
.....2514½ Thirteenth, Columbus, Nebr., **OALR**
Campbell, Duncan A., '19.....
.....David Whitney bldg., Detroit, Mich., **OALR**
Campbell, Edward H., '38.....
Lt. Comdr. (M.C.), 2035 Delancey, Philadelphia, Pa., **ALR**
Campbell, Glenn S., '37, Comdr. (M.C.), U. S. Navel Med.
Supply Depot, Pearl and Sands sts., Brooklyn, N. Y., **ALR**
Campbell, Malcolm D., '27, Army Air Corps,
.....Mantell Plaza Hotel, Miami Beach, Fla., **OALR**
Campbell, Paul A., '39, Lt. Col. (M.C.),
School of Aviation Med., Randolph Field, Tex., **ALR**
Campbell, Sherburne, '36, Lt. Comdr. (M.C.),
U. S. Naval Hosp., Philadelphia, Pa., **OP**
Campbell, W. K., '19..96 Third av., Long Branch, N. J., **OALR**
Campbell, William, '39.....
.....144 S. Harrison, East Orange, N. J., **ALR,**
Campbell, Wm. E., Jr., '36.....
.....Medical Arts bldg., Atlanta, Ga., **OP**
Canfield, Norton, '33, Lt. Col. (M.C.),
789 Howard av., New Haven, Conn., **ALR**
Capeles, Thomas Francis, '42.....
.....191 Merrimack st., Haverhill, Mass., **ALR**
Cardwell, Edgar P., '29, Maj. (M.C.), A.A.F.T.T.C.,
Basic Training Center No. 6, St. Petersburg, Fla., **ALR**
Carelli, Paul Vincent, '41.....
.....3626 W. Chicago av., Chicago, Ill., **OP**
Carl, Evan Elsworth, '40.....
.....606 West Wisconsin av., Milwaukee, Wis., **OP**
Carmody, Raymond F., '37...504 Broadway, Gary, Ind., **OP**
Carmody, T. E., '04...Metropolitan bldg., Denver, Colo., **ALR**
Caron, Armand L., '39...36 Pleasant, Worcester, Mass., **ALR**
Carpenter, Chapin, '30..1930 Chestnut, Philadelphia, Pa., **OP**
Carrasquillo, Honorio Fernando, '41.....
.....P. O. Box 128, Utuado, Puerto Rico, **OP**
Carroll, Frank D., '37...635 W. 165th, New York, N. Y., **OP**
Also: Blind Brook Lodge, Rye, N. Y.,
Carroll, Walter J. E., '37...5 Chestnut, Arlington, Mass., **ALR**
Carruth, Howard E., '28.....
.....Medical Arts bldg., Portland, Ore., **OALR**
Carson, W. E., '17.....Jenkins Arcade, Pittsburgh, Pa., **OP**
Carter, Albert A., '37.....419 Boylston, Boston, Mass., **OP**
Carter, E. D., '30.....Central bldg., Wichita, Kan., **ALR**
Carter, John M., '29.....
.....David Whitney bldg., Detroit, Mich., **ALR**
Carter, Leland F., '37.....
.....David Whitney bldg., Detroit, Mich., **OP**
Carter, W. W......6 Chestnut av., Bronxville, N. Y., **ALR**
Caruthers, Samuel B., '37.....
.....Grenada Clinic, Grenada, Miss., **OALR**
Cary, E. H., '12.....Medical Arts bldg., Dallas, Texas, **OALR**
Cary, G. C., '18....Canon Block, Grand Junction, Colo., **OALR**
Case, Paul Henry, '39...15 E. Monroe, Phoenix, Ariz., **OP**
Cassady, J. Vernal, '28.....
.....Sherland bldg., South Bend, Ind., **OALR**
Cassidy, Waldron A., '31.....
.....Medical Arts bldg., Omaha, Nebr., **ALR**
Casten, Virgil G., '32, Lt. Comdr. (M.C.),
520 Commonwealth av., Boston, Mass., **OP**
Castroviejo, Ramon, '31.....
.....635 W. 165th, New York, N. Y., **OP**
Cave, Linus S., '29..State Tower bldg., Syracuse, N. Y., **ALR**
Cawthorne, Harold Jesse, '41.....
.....239 Pipestone, Benton Harbor, Mich., **ALR**
Cecha, Albin H., '35, c/o Mrs. Nancy Cecha,
4437 Woolworth av., Omaha, Nebr., **OP**

Chadwick, Ira B., '20..717½ Walnut, Coffeyville, Kan., **OALR**
Chaffers, Wm. H., '31.....190 Bates, Lewiston, Maine, **ALR**
Chainski, Edward L., '32....130 N. Wells, Chicago, Ill., **ALR**
Chamberlain, Webb P., '31.....
.....**7405 Detroit av., Cleveland, Ohio, OP**
Champion, Albert N., '36.....
.....Medical Arts bldg., San Antonio, Texas, **ALR**
Chandler, Arthur Cecil, '42, Capt. (M.C.),
1562 Virginia st., Charleston, W. Va., **OP**
Chandler, G. E., '30....Huntington bldg., Miami, Fla., **ALR**
Chandler, Paul Austin, '38.....
.....5 Bay State Road, Boston, Mass., **OP**
Chapman, Katharine H., '36.....
.....304 Burns bldg., Colorado Springs, Colo., **OP**
Chapman, S. Jefferson, '26.....
.....Burns bldg., Colorado Springs, Colo., **ALR**
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...435 N. Palm Canyon drive, Palm Springs, Calif.,
and 324 E. Wisconsin av., Milwaukee, Wis., **OALR**
Chappell, R. S., '25.....
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Charles, Joseph W., '22..Humboldt bldg., St. Louis, Mo., **OP**
©Charlton, C. Coulter, '16.....
.....124 S. Illinois av., Atlantic City, N. J., **ALR**
Chase, Carl Clarence, '39..121 Main, Middletown, Conn., **OP**
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Chase, S. B., '25.....Carver bldg., Ft. Dodge, Iowa, **OALR**
Chase, W. D., '24.....230 E. Broad, Bethlehem, Pa., **ALR**
Cheek, Carey, '38...Medical Arts bldg., Springfield, Mo., **OP**
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.....377 Commonwealth av., Boston, Mass., **ALR**
Cheney, James W., '20..Schweiter bldg., Wichita, Kan., **OALR**
Cheney, Ralph E., '36...United Life bldg., Salina, Kan., **OP**
Chenoweth, Charlie Everette, '42.....
.....Forester's bldg., Mason City, Iowa, **ALR**
Chew, William Morgan, '38, Lt. Comdr. (M.C.), U. S.
Naval Academy, Bancroft Hall, Annapolis, Md., **ALR**
Chew, William Morgan, '38, Lt. Comdr., U. S. Naval
Academy, Bancroft Hall, Annapolis, Md., **ALR**
Childers, Robert J., '29....604 Park av., Plainfield, N. J., **ALR**
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Chipman, L. DeV., '25..42 Coburg, St. John, N. B., Can., **OALR**
Chisholm, Julian F., Jr., '36.....
.....20 Phillips st., Boston, Mass., **OP**
Christoph, Carl H., '28..30 N. Michigan av., Chicago, Ill., **OALR**
Cinelli, Albert A., '36.....101 E. 85, New York, N. Y., **ALR**
Clark, Cecil P., '31.....23 E. Ohio, Indianapolis, Ind., **OP**
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Dumas Edward DeVan, '36..... .....Medical Arts bldg., San Antonio, Texas, **ALR**
Dunaway, Carl E., '35 ..Huntington bldg., Miami, Fla., **ALR**
Dunbar, John Charles, '40. Empire bldg., Pittsburgh, Pa., **OP**
Duncan, Perry E., '37...101½ E. Main, Taylorville, Ill., **OP**
Duncan, R. A., '29.....Amarillo bldg., Amarillo, Texas, **ALR**
(H)Dundas-Grant, Sir James (Retired)..... England,
Dunlap, Henry A., '37..... .....7815 E. Jefferson av., Detroit, Mich., **OP**
Dunlap, Lawrence G., '20..101 Main, Anaconda, Mont., **OALR**
Dunn, Austin G., '37..... .....111½ Strongs av., Stevens Point, Wis., **ALR**
Dunnavan, Floyd Leighton, '42..... .....Arts bldg., Vancouver, Wash., **OP**
Dunnington, J. H., '24...30 W. 59th, New York, N. Y., **OP**
Dunphy, Edwin B., '31..... ..Lt. Comdr. (M.C.), 243 Charles st., Boston, Mass., **OP**
(L)Duntley, G. S., '13.....Box 47, Macomb, Ill., **OALR**
Durr, S. A., '27..Medico-Dental bldg., San Diego, Calif., **OP**
(S)Dutrow, H. V., '14, Maj. (M.C.), A.P.O. 845, c/o Postmaster, New York, N. Y., **OALR**
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Dyer, C. P., '20.....Humboldt bldg., St. Louis, Mo., **OP** and Garelock bldg., Webster Groves, Mo.,
Dykman, Augustus E. '37..... .....833 S. W. 11th av., Portland, Ore., **OP**
Dysart, Ben Robnett, '41..... .....65 N. Madison av., Pasadena, Calif., **ALR**

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Ⓑ Elliot, R. H., '13, Lt. Col., 54 Welbeck st., Cavendish sq. W., London, Eng.,

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F

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Ferguson, George Burton, '40 ..... 1114 West Main st., Durham, N. C., **ALR**

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Fernandez, Luis J., '40, Institute Oftalmico de Puerto Rico, San Juan, Puerto Rico, **OP**

Fernandez, Ricardo F., '39 ..... 15 Ponce de Leon av., San Juan, Puerto Rico, **OP**

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Filmer, George Arnold, '41, Capt. (M.C.), William Beaumont General Hospital, El Paso, Tex., **OP**

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Fink, Otto E., '38 ..... 9 W. Madison, Danville, Ill., **OP**

Fink, Walter H., '27 ..... Medical Arts bldg., Minneapolis, Minn., **OP**

Finney, Nancy E., '37..2525 Gilbert av., Cincinnati, Ohio, **OP**
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Fischbach, V. W., '31.....
.....Union Central bldg., Cincinnati, Ohio, **ALR**
Fischer, Abraham, '39..Mercantile bldg., Rochester, N.Y., **OP**
Fischer, Aubrey, '34.....
.....1835 Eye st. N.W., Washington, D. C., **ALR**
Fischer, N. Arthur, '26.....
.....Union Trust bldg., Pittsburgh, Pa., **ALR**
(B)Fischer, V. B., '17..Mercantile Bank bldg., Boulder, Colo., **OALR**
(D)Fisher, Carl, '12..1930 Wilshire blvd., Los Angeles, Calif., **OP**
Fisher, George Garthwaite, '41.....
.....271 New York av., Brooklyn, N. Y., **ALR**
Fisher, Gilbert Eugene, '42.....
.....Medical Arts bldg., Birmingham, Ala., **ALR**
Fisher, Guy Rothwell, '42.....
.....1 E. Beverley st., Staunton, Va., **ALR**
Fisher, H. Noland, '37.....
.....Olney Sanatorium Clinic, Olney, Ill., **OP**
Fisher, James A., '25.....
.....501 Grand av., Asbury Park, N. J., **OALR**
Fisher, Lawrence Christian, '41.....
.....469 W. Market, York, Pa., **OP**
(D)Fisher, W. A., '96.....30 N. Michigan av., Chicago, Ill., **OALR**
Fishman, Louis Z., '35.....
.....30 N. Michigan blvd., Chicago, Ill., **ALR**
Fishof, Frank E., '41.....
.....860 Grand Concourse, New York, N. Y., **ALR**
(D)Fiske, George F., '03...25 E. Washington, Chicago, Ill., **OALR**
Fitzgerald, Fred J. C., '34..118 E. 65th, New York, N. Y., **OP**
Fitzgerald, J. P., '25...30 N. Michigan av., Chicago, Ill., **OP**
Fitzgerald, William W., '37..27 Ludlow, Yonkers, N. Y., **OP**
Fitz-Hugh, Glassell S., '39, Capt. (M.C.),
...Wm. Beaumont General Hospital, El Paso, Tex., **ALR**
Fitzhugh, William McP., Jr., '38.....
Lt. Comdr. (M.C.), 384 Post, San Francisco, Calif., **ALR**
Flack, J. Vincent, '37, Lt. Comdr. (M.C.),
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Flaherty, Norman W., '39.....
.....10593 W. Jefferson av., River Rouge, Mich., **OP**
Flanders, Merton Newcomb, '42.....
.....Tilton General Hospital, Fort Dix, N. J., **ALR**
(B)Flatley, T. J., '14.....1518 Fifth av., Moline, Ill., **OALR**
Flynn, Charles T., '27..41 Thumbull, New Haven, Conn., **ALR**
Flynn, James A., '24.....
...1511 Rhode Island av. N.W., Washington, D. C., **ALR**
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Naval Air Station (Dispensary), Pasco, Wash., **ALR**
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Folken, Frank G., '28.....
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Station, Medical Arts bldg., San Juan, Puerto Rico, **ALR**
Forbes, Sherman B., '22.....706 Franklin, Tampa, Fla., **OALR**
Ford, Hanby L., '28.....Robeson bldg., Champaign, Ill., **OALR**
Ford, Leroy Sargent, '42.....30 School st., Keene, N. H., **ALR**
Ford, William W., '38..109 E. Walnut, Green Bay, Wis., **ALR**
Forsythe, S. T., '19.....Rose bldg., Cleveland, Ohio, **OALR**
Foster, Clarence Batchelder, '42, Capt. (M.C.),
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Foster, George C., '36.....807 Broadway, Fargo, N. D., **OALR**
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Foster, Harold, '35.....
....92-11 35th av., Jackson Hts., New York, N. Y., **ALR**
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Foster, Warren H., '35..217 Fifth av. S., Clinton, Iowa, **ALR**
Foster, Wayne J., '35..Higley bldg., Cedar Rapids, Iowa, **ALR**
Fowler, Earl B., '25.....55 E. Washington, Chicago, Ill., **OP**
Fowler, Edmund Prince, Jr., '40.....
.....180 Fort Washington av., New York, N. Y., **ALR**
Fowler, James G., '37..412 Linwood av., Buffalo, N. Y., **OP**
Fowler, Robert H., '29, Maj. (M.C.),
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(D)Frank, Ira, '05.....104 S. Michigan av., Chicago, Ill., **ALR**
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Fraser, Robert C., '29..940 Military, Port Huron, Mich., **ALR**
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Freimuth, L. E., '27.....916 S. Flood, Norman, Okla., **ALR**
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French, Robert W., '37....181 Pleasant, Malden, Mass., **OP**
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Friedman, Elmer Arthur, '41.....
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Gilbert, Joseph G., '36, Lt. Comdr. (M.C.), Naval Hosp.,
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Gilbert, Ralph H., '41 Metz bldg., Grand Rapids, Mich., **OP**
Gilfillan, Homer John, Jr., '41 1416 Main, Quincy, Ill., **ALR**
Gill, E. G., '19 711 S. Jefferson, Roanoke, Va., **ALR**
Gill, E. King, '36, Maj. (M.C.), Mators Army Air Field,
Greenville, Tex., **ALR**
Gill, G. Bache, '23
1726 Eye st. N.W., Washington, D. C., **ALR**
Gill, Wm. Davis, '29
705 E. Houston, San Antonio, Texas, **OALR**
Gillespie, Elmer H., '38 1101 Beacon, Brookline, Mass., **ALR**
Gillett, Wilbur G., '20 201½ N. Main, Wichita, Kan., **OP**
Gillette, D. F., '25 State Tower, Syracuse, N. Y., **OP**
Gilligan, Alexander, '39 1154 Dean, Brooklyn, N. Y., **ALR**
Gilliland, O. S., '30 Professional bldg., Kansas City, Mo., **ALR**
Gillum, J. R., '20 221 S. 6th, Terre Haute, Ind., **OALR**
Gilmore, Goodlatte Browne, '41
2940 Grand Concourse, New York, N. Y., **ALR**
Gilmore, Milton A., '36 Box 172, Parkersburg, W. Va., **OP**
Gilmore, Robert O., '39 1159 Hancock, Quincy, Mass., **ALR**
Gipner, J. F., '26 Medical Arts bldg., Rochester, N. Y., **OP**
Gittins, T. R., '20 Davidson bldg., Sioux City, Iowa, **OALR**
Givner, Isadore, '36 108 E. 66th, New York, N. Y., **OP**
Glasford, S. T., '24 416 Elizabeth, Pekin, Ill., **OALR**
Glatt, Morris A., '31 55 E. Washington, Chicago, Ill., **ALR**
Gleason, John E., '40
David Whitney bldg., Detroit, Mich., **ALR**
Glick, Harry N., '28 3115 S. Grand blvd., St. Louis, Mo., **ALR**
(L)Glosser, H. H., '12 448 Franklin, Buffalo, N. Y., **OP**
Glover, Arthur C., '33 311 W. Church, Elmira, N. Y., **OALR**
Glover, Lewis P., '38 1200 Fourteenth av., Altoona, Pa., **OP**
Glowacki, Ben F., '28 Maccabees bldg., Detroit, Mich., **OALR**
Glushak, Leopold, '38 1 West 85th, New York, N. Y., **ALR**
Goad, R. R., '31, Lt. Comdr. (M.C.),
4211 Farragut av., Hyattsville, Md., **OP**
Goar, Everett L., '26 1300 Walker av., Houston, Texas, **OP**
(L)Goddard, H. M., '11 1531 Spruce, Philadelphia, Pa., **ALR**
Goetzman, Arthur Cecil, '42
8907 Hennepin av., Niagara Falls, N. Y., **OP**
Goehring, Donald Everett, '41
Butler S. & T. bldg., Butler, Pa., **ALR**

Goldbach, L. J., '11.....6 East Eager, Baltimore, Md., **OP**
Goldberg, Herman Krieger, '42.....
—.—.—.....8 E. Eager st., Baltimore, Md., **OP**
Goldberg, Sol, '39, Maj. (M.C.), La Garde Gen. Hosp., New Orleans, La., **OP**
Goldberg, Sylvan S., '35.....
—.....1917 Wilshire blvd., Los Angeles, Calif., **ALR**
Goldcamp, Edward C., '32.....
.....Dollar Bank bldg., Youngstown, Ohio, **ALR**
Goldcamp, John Stephen, '41, Capt. (M.C.), 810 Dollar Bank bldg., Youngstown, Ohio, **OP**
Golden, Matthew G., '33..1 Nevins st., Brooklyn, N. Y., **ALR**
Goldenburg, Michael, '31.....
—.....104 S. Michigan av., Chicago, Ill., **OP**
Golding, Joseph E., '25.....1 Nevins, Brooklyn, N. Y., **OP**
Goldman, A. Milton, '29.....
....1 N. Village av., Rockville Centre, L. I., N. Y., **OP**
Goldman, H. G., '23..57 W. 58th st., New York, N. Y., **ALR**
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—.....121 E. 60th, New York, N. Y., **ALR**
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Maj. (M.C.), Station Hospital, Camp Blanding, Fla., **OP**
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.....Medical Arts bldg., Toronto, Can., **ALR**
Goldstein, Henry Z., '36, Lt. (M.C.), Induction Center, Rochester, N. Y., **ALR**
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Gollom, Joseph, '38....425 Bloor st. W., Toronto, Can., **ALR**
Gomon, Louis D., '37.....Eddy bldg., Saginaw, Mich., **OP**
Gonne, Wm. S., '36.....
.....David Whitney bldg., Detroit, Mich., **ALR**
Gooch, John B., '35..Canal Bank bldg., New Orleans, La., **ALR**
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.....Capt. (M.C.), Hunter Field, Savannah, Ga., **OALR**
Good, Richard Peterson, '41.....
Maj. (M.C.), Station Hospital, Fort Brady, Mich., **ALR**
Good, Wm. M., '29.....63 Center, Waterbury, Conn., **ALR**
Goodale, Robert L., '36..330 Dartmouth, Boston, Mass., **ALR**
Goodall, Edwin B., '34.....
.....71 Bay State road, Boston, Mass., **OP**
Goode, Edward A., '29.....
.....143—28 Sanford av., Flushing, N. Y., **ALR**
Goodell, William, '34..121 Chestnut, Springfield, Mass., **ALR**
Goodfellow, Thomas J., '39.....
.....Arcade bldg., Saratoga Springs, N. Y., **OALR**
Goodhill, Victor, '42, 1101 Wilshire Medical bldg., 1930 Wilshire blvd., Los Angeles, Calif., **ALR**
Goodman, Edgar L., '36.....
.....1801 Eye st. N.W., Washington, D. C., **OP**
Goodman, Thomas L., '31.....
.....1601 Clover Lane, Fort Worth, Tex., **ALR**
Goodspeed, Harry B., '34.....
.....36 Pleasant st., Worcester, Mass., **ALR**
Goodwin, Thomas M., '38..Golden Clinic, Elkins, W. Va., **ALR**
Goodyear, H. M., '24...Doctors bldg., Cincinnati, Ohio, **ALR**
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.....1616 Pacific av., Atlantic City, N. J., **OP**
Gordon, Charles H., '26...46 Deering, Portland, Maine, **ALR**
Gordon, Glenn G., '29.....
.....1227 E. Ocean blvd., Long Beach, Calif., **ALR**
Gordon, J. S., '19.....Bankers bldg., Milwaukee, Wis., **OALR**
Gordon, William, '33.....1721 Pine, Philadelphia, Pa., **ALR**
Gorin, Meyer, '42....35 Chestnut st., Rochester, N. Y., **ALR**
Gorman, James R., '36.....
.....Allied Arts bldg., Lynchburg, Va., **ALR**
Gorney, Arthur J., '39.....
.....475 Commonwealth av., Boston, Mass., **ALR**

Gorrell, T. J. H., '09.....
.....16th and Vincennes, Chicago Heights, Ill., **OALR**
Gorilla, Laurence Vincent, '42.....
.....3115 S. Grand blvd., St. Louis, Mo., **OP**
Gosney, C. W., '20.....
.....5217 Hollywood blvd., Los Angeles, Calif., **OP**
Goss, Harold L., '37.....4502 E. 33rd, Seattle, Wash., **OP**
Gottlieb, Bernard N., '39.....
.....387 Eastern Parkway, Brooklyn, N. Y., **ALR**
Gough, Roy H., '31....600 W. 10th, Fort Worth, Texas, **OALR**
Gould, Nathaniel Jay, '37.....
.....940 Grand Concourse, New York, N. Y., **OP**
Gouterman, J. I., '30, Maj. (M.C.), 344 W. School Lane (Germantown), Philadelphia, Pa., **OP**
Goux, Raymond S., '22.....
.....David Whitney bldg., Detroit, Mich., **OALR**
Gowen, George R., '29..Baker bldg., Walla Walla, Wash., **ALR**
Gowen, Thomas F., '39, Naval Dispensary, Navy Dept. bldg., 194 Constitution av., Washington, D. C., **ALR**
Gowens, Henry Lytle, Jr., '42.....
.....123 S. 23rd st., Philadelphia, Pa., **OP**
Grace, Charles C., '33..145 King st., St. Augustine, Fla., **ALR**
Gracey, George F., '24.....
.....209 State, Harrisburg, Pa., **OALR**
Gradle, Harry S., '11...58 E. Washington, Chicago, Ill., **OP**
Graebner, Herbert, '34.. 45 Park av., New York, N. Y., **ALR**
Graef, Fred W., '36.....850 Park av., New York, N. Y., **ALR**
Granberry, Carl, '34.....
.....Maison Blanche bldg., New Orleans, La., **ALR**
Granger, W. B., '20.....Gazette bldg., Emporia, Kan., **OALR**
Grant, Fred E., '12.....
...Kalamazoo Nat'l Bank bldg., Kalamazoo, Mich., **OALR**
Grant, H. W., '30.....
.....Lowry Med. Arts bldg., St. Paul, Minn., **OP**
Grant, Maurice David, '41.....
.....1860 Morris av., New York, N. Y., **ALR**
Graves, Basil, '26.....
11 Devonshire pl., Upper Wimpole, W.1, London, Eng.,
Grecar, J. N., Jr., '29.....
.....1740 M st. N.W., Washington, D. C., **OP**
Green, Hyman S., '35, Lt. Comdr. (M.C.), Naval Training Station, Great Lakes, Ill., **OALR**
Green, John, '05 3720 Washington blvd., St. Louis, Mo., **OP**
Greenberg, Maynard M., '39, Capt. (M.C.), Station Hospital, Camp Crowder, Mo., **OP**
Greene, James S., '36.. 61 Irving pl., New York, N. Y., **ALR**
Greene, Laurence W., '28..Republic bldg., Denver, Colo., **ALR**
Greene, M. L., '30.....
.....Missouri Theatre bldg., St. Louis, Mo., **OP**
Greenfield, Samuel D., '27.....
.....169 New York av., Brooklyn, N. Y., **ALR**
Greenfield, William J., '28.....
.....50 Anderson, Hackensack, N. J., **ALR** and 777 West End av., New York, N. Y.,
Greenlee, Max Russell, '38, Lt. Comdr. (M.C.), U. S. Naval Hospital, Farragut, Idaho, **ALR**
Greenwood, G. J., '30..55 E. Washington, Chicago, Ill., **ALR**
Greever, B. L., '20.....100 N. Main, Hutchinson, Kan., **OALR**
Gregg, John B., '20.....
.....Security Nat'l Bank bldg., Sioux Falls, S. D., **OALR**
Gresser, Edward B., '29..39 E. 75th, New York, N. Y., **OP**
Griesman, Bruno L., '39.....
.....1100 Park av., New York, N. Y., **ALR**
Griffey, Edward W., '29.....
.....Medical Arts bldg., Houston, Texas, **OALR**
Griffey, Walter P., '39.....
.....Marine Hosp. Stapleton, New York, N. Y., **OP**

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18 Prospect Park West, Brooklyn, N. Y., **ALR**
Griffith, Charles M., '36
Comdr. (M.C.), 140 E. 54th st., New York, N. Y., **ALR**
Griffith, H. M., '30
Johnstown Trust bldg., Johnstown, Pa., **OP**
Grigsby, Roll O., '40
100 E. Second st., Ashland, Wis., **ALR**
Grimaldi, Americo John, '40
100 West 59th st., New York, N. Y., **OP**
Grimmett, Roger Skelton, '40
217 Park Place, Ashtabula, Ohio, **ALR**
Griscom, J. Milton, '20
255 S. 17th, Philadelphia, Pa., **OP**
Griswold, George W., '35
211 W. 3rd, Roswell, N. M., **ALR**
Gross, Julius E., '20
3401 Fifth av., Pittsburgh, Pa., **ALR**
Grossman, Herman P., '38
210 Angell, Providence, R. I., **OP**
Grove, Bruce Alexander, '42
141 E. Market st., York, Pa., **OP**
Grove, Russell C., '37
60 E. 58th, New York, N. Y., **ALR**
(S)Grove, W. E., '14
324 E. Wisconsin av., Milwaukee, Wis., **OALR**
Gruppe, Karl W., '38, Maj. (M.C.),
Army Flying School, Lawrenceville, Ill., **ALR**
(L)Grushlaw, Israel, '05
225 W. 86th, New York, N. Y., **ALR**
Gsell, George Franklin, '38
In Service, Lowry Field, Denver, Colo., **OP**
Gsell, J. F., '20
911 Beacon bldg., Wichita, Kan., **OALR**
Gubner, Julius, '37
25 Lefferts av., Brooklyn, N. Y., **ALR**
(S)Guggenheim, L. K., '18
1035 Gayley av. West Los Angeles, Calif., **ALR**
Gulbor, George P., '33
30 N. Michigan av., Chicago, Ill., **OP**
Gulick, John Duncan, '41
704 Union, Schenectady, N. Y., **ALR**
Gump, Millard E., '39
In Service, 411 Thirtieth, Oakland, Calif., **OP**
(L)Gundelach, C. A., '12
University Club bldg., St. Louis, Mo., **ALR**
Gunderson, Trygve, '35
Maj. (M.C.), 5 Bay State Road, Boston, Mass., **OP**
Gundrum, Lawrence Kramer, '42
1930 Wilshire blvd., Los Angeles, Calif., **ALR**
Gunn, M. L., '29
Metcalf bldg., Harlan, Ky., **ALR**
Gurtov, John J., '37
1235 Grand Concourse, New York, N. Y., **ALR**
Gustafson, Olga Frideberg, '38
185 N. Wabash av., Chicago, Ill., **OP**
Guttman, M. Reese, '29
30 N. Michigan av., Chicago, Ill., **ALR**
Guy, Loren P., '34
40 E. 62nd, New York, N. Y., **OP**
(S)Guyton, B. S., '18
512 Van Buren av., Oxford, Miss., **OALR**
Guyton, Jack Smallwood, '41
Johns Hopkins Hospital, Baltimore, Md., **OP**

H

Haas, Louis Richard, '42
Smith Clinic, Pittsburg, Kan., **OALR**
(L)Haden, Henry C., '13
1914 Travis, Houston, Texas, **OP**
Haessler, F. Herbert, '29
Wells bldg., Milwaukee, Wis., **OP**
Hagaman, Van Dyke, '34
Lamar Life bldg., Jackson, Miss., **ALR**
Hagan, Cornelius Edward, Jr., '42
Capt. (M.C.), 31 Evergreen av., Lynbrook, N. Y., **ALR**
Hageman, George Ricker, '42
Longmont Hospital, Longmont, Colo., **ALR**
Hagemann, John A., '25
Highland bldg., Pittsburgh, Pa., **ALR**
Hagens, Elmer William, '41
30 N. Michigan, Chicago, Ill., **ALR**
Haggart, Harry H., '37
Carew Tower, Cincinnati, Ohio, **ALR**
Hague, Eliott B., '39
454 Franklin, Buffalo, N. Y., **OP**
Hahn, Wm. H., '30
15 Lombardy, Newark, N. J., **OP**
Hahn, William N., '38
City Nat'l Bk. bldg., Omaha, Nebr., **OP**
Haire, Paul G., '35
6 Pleasant st., Malden, Mass., **OP**
Hale, Arthur S., '28
Eaton Tower, Detroit, Mich., **OALR**
Haley, Paul J. D., '37
333 Winthrop st., Medford, Mass., **ALR**
Haley, Wm. T., '33
531 Beacon, Boston, Mass., **ALR**
Hall, Alice K., '24
25 E. Washington, Chicago, Ill., **ALR**
Hall, Colby, '42, Capt. (M.C.), 73rd Evacuation Hosp.,
National Military Home, Los Angeles, Calif., **ALR**
Hall, Edwin P., '28
76 Chestnut, Oneonta, N. Y., **ALR**
(L)Hall, G. C., '07
Brown bldg., Louisville, Ky., **OALR**
Hall, Lawrence H., '39
1520 Oak st., South Pasadena, Calif., **OALR**
Hall, Raymond A., '39, Capt. (M.C.),
3521 Hopkins Road, Youngstown, Ohio, **ALR**
Hall, Sobisca S., '34
Maj. (M.C.), Station Hospital, Keesler Field, Miss., **ALR**
Hallberg, Olav Erik, '42
Mayo Clinic, Rochester, Minn., **ALR**
Hallum, Alton V., '34
478 Peachtree, Atlanta, Ga., **OP**
(S)Halsted, Fred S., '18
Metropolitan bldg., Denver, Colo., **OALR**
Halton, Edward P., '37
245 Maple, Holyoke, Mass., **ALR**
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10515 Carnegie av., Cleveland, Ohio, **ALR**
Hamilton, Richard G., '40
Medical Arts bldg., Pittsburgh, Pa., **ALR**
Hamlin, Fred E., '26
Medical Arts bldg., Roanoke, Va., **ALR**
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David Whitney bldg., Detroit, Mich., **ALR**
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May bldg., Pittsburgh, Pa., **ALR**
Hamrick, DeWitt, '40
601 Jackson, Corinth, Miss., **ALR**
Hanckel, Richard White, Jr., '42
96-A Bull st., Charleston, S. C., **ALR**
Hancox, Cecil W., '39
Bahr Arcade bldg., Pottstown, Pa., **ALR**
Hand, Frank, '35
450 Sutter, San Francisco, Calif., **ALR**
Hands, Sidney G., '26
201 W. 2nd, Davenport, Iowa, **ALR**
Haney, Wm. P., '20
Medical Arts bldg., Omaha, Nebr., **OALR**
Hankins, George Geddy, '41
Medical Arts bldg., Newport News, Va., **ALR**
Hanley, James S., '37
40 W. 55th, New York, N. Y., **ALR**
Hanley-Roeling, Jeanne C., '39
Maison Blanche bldg., New Orleans, La., **ALR**
Hannon, Robert Emmet, '42, Capt. (M.C.), A.U.S.,
Oklahoma City Air Depot, Oklahoma City, Okla., **OP**
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Provident Bank bldg., Cincinnati, Ohio, **OALR**
Hansel, French K., '26
634 N. Grand av., St. Louis, Mo., **ALR**
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Medical Arts bldg., Minneapolis, Minn., **OALR**
Hanson, Wm. L., '29
Spivey bldg., East St. Louis, Ill., **ALR**
Hantman, Irvin, '39
Farragut Medical bldg., Washington, D. C., **ALR**
Happ, Linley Conrad, '41
280 Beacon st., Boston, Mass., **ALR**
Hara, H. James, '33
Hinsdale Sanitarium and Hospital, Hinsdale, Ill., **ALR**
Haralson, Charles H., '29
Medical Arts bldg., Tulsa, Okla., **OP**
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135 N. Main, Bellefontaine, Ohio, **OALR**
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Missouri Theatre bldg., St. Louis, Mo., **OP**
Hardie, G. C., '30
290 W. Michigan av., Jackson, Mich., **OALR**
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Hume-Mansur bldg., Indianapolis, Ind., **OP**
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948 "G" av., Coronado, Calif., **OALR**

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Hardy, Walter B., '36. 2121 Highland, Birmingham, Ala., **ALR**
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.....17 Schermerhorn, Brooklyn, N. Y., **OP**
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...Merion and Montgomery aves., Bryn Mawr, Pa., **ALR**
Harkins, William Blake, '41, Lt. (M.C.), Co. A, Med.
Battalion, 9th Marine Reg., Camp Pendleton, Calif., **ALR**
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Station Hospital, Hill Field, Ogden, Utah, **ALR**
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.....Putnam bldg., Davenport, Iowa, **OALR**
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.....Maj. (M.C.), A.A.F.T.S., Madison, Wis., **ALR**
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U. S. Naval Hosp., Pearl Harbor, T. H., **OALR**
Harnick, Israel, '39. .1132 Delaware av., Buffalo, N. Y., **ALR**
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.....Reynolds bldg., Winston-Salem, N. C., **ALR**
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Naval Training Station, San Diego, Calif., **ALR**
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.....Hume-Mansur bldg., Indianapolis, Ind., **OP**
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.....Johnstown Trust bldg., Johnstown, Pa., **OALR**
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Station Hospital, March Field, Calif., **ALR**
Harris, H. B., '10. Fidelity Medical bldg., Dayton, Ohio, **OALR**
Harris, Harold Earl, '42.....
.....Cleveland Clinic, Cleveland, Ohio, **ALR**
Harris, Herbert H., '39, Capt. (M.C.),
Mellie Esperson bldg., Houston, Texas, **ALR**
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...Methodist Hosp Doctors bldg., Memphis, Tenn., **ALR**
Harris, Morris L., '40....20 Plaza st., Brooklyn, N. Y., **ALR**
Harris, Robin, '23.....964 N. State, Jackson, Miss., **ALR**
Harris, W. C., 1900.....19 W. 8th, Cincinnati, Ohio, **ALR**
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.....Second Nat'l Bank bldg., Connellsville, Pa., **OALR**
Hartenbower, George E., '38.....
.....Unity bldg., Bloomington, Ill., **ALR**
Hartgraves, Hallie, '38.....
.....28 West Adams av., Detroit, Mich., **OP**
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Hartman, Eugene C., '33.....
.....610½ Market, Parkersburg, W. Va., **OALR**
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Hartzell, Sol M., '11.....
.....260 Tod Lane, Youngstown, Ohio, **OALR**
Harvey, N. Darrell, '04.....
.....112 Waterman, Providence, R. I., **OALR**
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Haskin, W. H., '13.....375 Park av., New York, N. Y., **ALR**
Hasty, F. E., '25...3631 West End av., Nashville, Tenn., **ALR**
Hatch, C. B., '08.....3 W. Church, Newark, Ohio, **OALR**
Hatcher, William F., '38.....
.....409 Medical Arts bldg., Roanoke, Va., **OP**
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Haughey, Wilfrid H., '11. Post bldg., Battle Creek, Mich., **OALR**
Hauser, I. Jerome, '38, Maj. (M.C.),
O'Reilly General Hospital, Springfield, Mo., **ALR**
Havens, Fred Z., '36....Mayo Clinic, Rochester, Minn., **ALR**
Hawkins, Raymond Willard, '40.....
.....39 S. Goodman, Rochester, N. Y., **ALR**
Hawman, Erle G., '20. 4051 N. Broad, Philadelphia, Pa., **OP**
Hawthorne, Allen T., '38.....
.....110 N. Braddock, Winchester, Va., **ALR**
Hayden, Jerome William, '42.....
.....4458 Madison st., Chicago, Ill., **ALR**
Hayes, Merrill B., '40.....
...Capt. (M.C.), Station Hospital, Ft. Belvoir, Va., **ALR**
Hayes, Robert T., '36. 42 Coburg, St. John, N. B., Can., **ALR**
Hays, Charles E., '39. U. S. Bank bldg., Johnstown, Pa., **ALR**
Hazen, S. Frank, '39.....231 Chestnut, Meadville, Pa., **ALR**
Heard, Mary K., '10.....
...R.F.D. 2, Box 75, St. Petersburg, Fla. (Retired), **OP**
Heare, Louis C., '39.....
Maj. (M.C.), Station Hospital, Camp Howze, Tex., **OP**
Heath, Parker, '24. David Whitney bldg., Detroit, Mich., **OP**
Hebble, Howard Miller, '40, Lt. (j. g.) (M.C.),
U. S. Naval Hospital, Philadelphia, Pa., **ALR**
Hebert, Arthur W., '27. 450 Sutter, San Francisco, Calif., **OALR**
Hedlund, Gerald O., '36, Lt. Comdr. (M.C.) U.S.N.R.,
N. S. Naval Training Station, Great Lakes, Ill., **ALR**
Heeb, Harry J., '18.....
.....212 W. Wisconsin av., Milwaukee, Wis., **OALR**
Heed, C. R., '10.....1205 Spruce, Philadelphia, Pa., **OP**
Heetderks, D. R., '30.....
.....Medical Arts bldg., Grand Rapids, Mich., **ALR**
Heidelman, George, '39.....
.....Union Central bldg., Cincinnati, Ohio, **OP**
Heinberg, Charles J., '31.....
.....Medical Center, Pensacola, Fla., **ALR**
Heine, Lyman H., '38....635 N. Main, Fremont, Nebr., **ALR**
Heine, William Howard, '41.....
.....Odd Fellow bldg., Fremont, Nebr., **ALR**
Heitger, J. D., '05.....Heyburn bldg., Louisville, Ky., **OALR**
Held, Albert E., '39, Maj. (M.C.), 37th General
Hospital Unit, Camp Blanding, Fla., **ALR**
Hempstead, Bert E., '20. Mayo Clinic, Rochester, Minn., **ALR**
Henderson, L. E., '30. 129 Clinton st., Watertown, N. Y., **OP**
Hendricks, Louis J., '31. Carew Tower, Cincinnati, Ohio, **OP**
Henner, Robert, '37.....
Capt. (M.C.), 1216 Third st. S.W., Ardmore, Okla., **ALR**
Hennessey, Wm. W., '20.....333 Essex, Salem, Mass., **OALR**
Henning, Carl, '08.....
..1752 Massachusetts av. N.W., Washington, D. C., **OP**
Henninger, L. L., '13.....
.....65 N. Madison av., Pasadena, Calif., **OALR**
Henry, George Arnold, '40, Capt. (R.C.A.M.C.),
Military Hospital, Camp Borden, Ont., Can., **ALR**
Henry, Robert, '42.....
...Capt. (M.C.), 322 E. James st., Colfax, Wash., **OP**
Hensle, Otto S., '40.....
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Kayser, Reuben, '39.....442 Bay Ridge Parkway. 75th st., Brooklyn, N. Y., **ALR**
Kazanjian, V. H., '33.....475 Commonwealth av., Boston, Mass., **LR**
Kearney, Harold L., '37.....1403 Delachaise, New Orleans, La., **ALR**
Kearny, R. A., '32.....2311 Connecticut av. N.W., Washington, D. C., **ALR**
Keating, T. F., '27.....Maccabee bldg., Detroit, Mich , **OALR**
Keefe, William J., '29.....205 E. Sidney av., Mt. Vernon, N. Y., **OALR**
Keeler, Lansing H., '34..89-04—148th st., Jamaica, N. Y., **OALR**
Keeler, Nelson S., '39....411 Thirtieth, Oakland, Calif., **ALR**
Keil, Frank C., '39...660 Madison av., New York, N. Y., **OALR**
Keil, Peter A., '36.....118 Pennsylvania av., Brooklyn, N. Y , **ALR**
Keim, W. F., '24.....25 Roseville av., Newark, N. J., **ALR**
Keiper, G. F., '30.....113 N. Church, Visalia, Calif., **ALR**
(S)Keith, W. E., '16..Professional bldg., Kansas City, Mo., **OALR**
Keller, S. A., '20.....303 S. Minnesota av., Sioux Falls, S. D., **OALR**
Kelley, Isaac D., Jr., '28.....3720 Washington blvd., St. Louis, Mo., **ALR**
Kelley, Vincent J., '33.....520 Commonwealth av., Boston, Mass., **ALR**
Kelly, Geo. F. J., '34.....37 S. 20th, Philadelphia, Pa., **OP**
Kelly, Joseph D., '28....140 E. 54th, New York, N. Y., **OALR**
Kemler, Joseph I., '22..1908 Eutaw pl., Baltimore, Md., **OALR**
Kemp, Roy C., '38.....343 State, Albany, N. Y., **ALR**
Kend, Lee, '32.....133 E. 58th, New York, N. Y., **ALR**
Kennedy, Eldred W., '28.....Gas & Electric bldg., Rochester, N. Y., **OP**
Kennedy, Patrick James, '40.....6910 Market st., Upper Darby, Pa., **OP**
Kennedy, Roscoe J., '39.....Lt. Comdr., 3277 Bradford Road, Cleveland, Ohio, **OP**
Kennedy, Wm. A., '36....372 St. Peter, St. Paul, Minn., **OP**
Kennon, W. G., '24....Doctors' bldg., Nashville, Tenn., **OALR**
Kenyon, Byron Benjamin, '42, Lt. Comdr. (M.C.), Naval Dispensary Submarine Base, New London, Conn., **ALR**
Kerns, Thomas C., '41.1110 West Main, Durham, N. C., **ALR**
Kershner, W. E., '34.....117 Front st., Bath, Maine, **OALR**
Kerst, John Arthur, '42.....Capt. (M.C.), R. R. No. 4, Springfield, Ill., **OP**
Kerwin, Raymond W., '38.....310 S. Michigan av., Chicago, Ill., **OALR**
Kessler, Mana, '38....311 Center av., Bay City, Mich., **ALR**
Kettelkamp, Fred O., '26.....Ferguson bldg., Colorado Springs, Colo., **ALR**
Key, Sam N., '26.....Norwood bldg., Austin, Texas, **OALR**
Keyes, John E. L., '33, Maj., 22199 McCauley road, Shaker Heights, Cleveland, Ohio, **OP**
Keys, Merton J., '22.....Pemberton bldg., Victoria, B. C., Can., **OALR**
Keyton, John A., '34.....304 N. Oates, Dothan, Ala., **ALR**
(S)Kiehle, F. A., '15.....1020 S. W. Taylor, Portland, Ore., **OALR**
Kilbourne, P. H., '24.....Fidelity Medical bldg., Dayton, Ohio, **OALR**
Kilgore, George L., '35, Maj. (M.C.), A.P.O. 923, Base Section 3, c/o Postmaster, San Francisco, Calif., **OP**
Kimbrig, Max, '40.....20 High st., Huntington, N. Y., **ALR**
(D)Kincaid, J. H., '03....421 Church av., Knoxville, Tenn., **OALR**
Kincannon, LeRoy T., '28.....1927 First av. N., Birmingham, Ala., **OALR**
King, Charles R., '20.....Spitzer bldg., Toledo, Ohio, **OALR**
King, Clair S., '37.....First Nat'l Bank bldg., Canton, Ohio, **OP**
King Edward, '21.....Carew Tower, Cincinnati, Ohio, **ALR**
King, George L., Jr., '29, Lt. Col. (M.C.), Gen. Disp. 2D201, Pentagon bldg., Washington, D. C., **OP**
King, Lowell W., '33.....470 E. State, Salem, Ohio, **ALR**
(S)King, O. H., '16...Medical Arts bldg., Hot Springs, Ark., **OALR**
King, Walter F., '38...519 Delaware av., Buffalo, N. Y., **OP**
Kinkade, Joseph M., '38.....359 E. Manchester blvd., Inglewood, Calif., **ALR**
Kinney, Charles Ed., '34.....10515 Carnegie av., Cleveland, Ohio, **ALR**
Kirby, D. B., '24.....780 Park av., New York, N. Y., **OP**
Kirkbright, James C., '35..40 E. 61st, New York, N. Y. **ALR**
Kirkpatrick, Hazen L., '37.....1320 Pembroke Lane, Topeka, Kan., **ALR**
(S)Kirkpatrick, Henry, '24.....Lt. Col., 54 Welbeck st., W. 1, London, Eng., **OP AR**
(S)Kistler, O. F., '18...43 N. Franklin, Wilkes-Barre, Pa., **OP**
(S)Kistner, F. B., '18.....Mayer bldg., Portland, Ore., **ALR**
Kleinert, Margaret N., '29.....82 Commonwealth av., Boston, Mass., **ALR**
Kleinfeld, Louis, '36.....17 E. 84th, New York, N. Y., **ALR**
Kler, Joseph H., '37.....151 Livingston av., New Brunswick, N. J., **ALR**
Klien, Bertha A., '38.....58 E. Washington, Sec. 2, Chicago, Ill., **OP**
(D)Kline, H. G., '12....State Tower bldg., Syracuse, N. Y., **ALR**
Kline, O. R., '30.....514 Cooper, Camden, N. J., **ALR**
(D)Klinedinst, John F., '06.....220 S. George, York, Pa., **OP A**
Klingner, Thomas O., '20.....Medical Arts bldg., Springfield, Mo., **OALR**
Klotz, William F., '38, Lt. Col. (M.C.), 168th Station Hospital, A.P.O. No. 948, Seattle, Wash., **ALR**
Kluever, Herman C., '36, Lt. Comdr. (M.C.), Carver bldg., Fort Dodge, Iowa, **OP**
(D)Knapp, Arnold, '08.....10 E. 54th, New York, N. Y., **OP**
Knapp, Arthur A., '33, Comdr. (M.C.), U. S. Naval Hospital, N.O.B., Norfolk, Va., **OP**
Knapp, F. N., '25....Medical Arts bldg., Duluth, Minn., **OALR**
Knauer, Wm. J., '30..Buckman bldg , Jacksonville, Fla., **OALR**
Knight, Granville F., '37.....900 North st., White Plains, N. Y., **ALR**
Knight, John S., '32.....Professional bldg., Kansas City, Mo., **ALR**
Knighton, Willis S., '31..121 E. 61st, New York, N. Y., **OP**

Knisely, Alan DeG., '38, Maj. (M.C.), Aviation Cadet Exam. Bd , 321 New P.O. bldg., Atlanta, Ga., **ALR**
Knopf, Saul, '37 ..... 30 E. 40th, New York, N. Y., **ALR**
Knowles, W. Mood, '39 ..... Medical Arts bldg., Dallas, Texas, **ALR**
Knowlton, Don S., '39 ..... Columbia Medical bldg., Washington, D. C., **ALR**
Kobrak, Heinrick G., '38, Capt. (M.C.), Station Hospital, Camp Adair, Ore., **ALR**
Koch, Ferdinand Leonard Philip, '40 ..... 408 St. Peter st., St. Paul, Minn., **OP**
Koebbe, Ezra E., '29, Lt. Comdr. (M.C.), U. S. Naval Hospital, Great Lakes, Ill., **ALR**
Koenig, Ivan J., '28 ... 40 W. North st., Buffalo, N. Y., **OP**
Kogut, Lewis Victor, '41, Capt. (M.C.), Erie Proving Grounds, LaCarne, Ohio, **OP**
ΦKoller, Carl, '34 ..... 68 E. 86th, New York, N. Y., **OP**
Koller, H. M., '36 ..... Medical Arts bldg., Minneapolis, Minn., **OP**
Koller, Louis R., 32 ..... Medical Arts bldg., Minneapolis, Minn., **ALR**
ΩKopetzky, S. J., '08 ..... 71 E. 80th, New York, N. Y., **ALR**
Kopp, Mortimer M., '42 ..... 79 Argyle road, Brooklyn, N. Y., **ALR**
Kornberg, Maurice, '38 ..... 441 E. 15th st., New York, N. Y., **ALR**
Kornzweig, Abraham L., '37 ..... 1200 Fifth av., New York, N. Y., **OP**
ΩKraft, Oscar H., '12 ..... 1518 N. Dearborn parkway, Chicago, Ill., **OP**
Krall, J. Thomas, '42 ..... Maj. (M.C.), 134 Lansdowne Court, Lansdowne, Pa., **OP**
Kramer, Julius C., '34...130 E. Market, Sandusky, Ohio, **ALR**
Krause, Arlington C., '37 ..... 950 E. 59th, Chicago, Ill., **OP**
ΩKrauss, Frederick, '10..1930 Chestnut, Philadelphia, Pa., **OALR**
Kravitz, Daniel, '40.... 861 Park place, Brooklyn, N. Y., **OP**
Kreag, Karl A., '39 ..... 167 Joralemon, Brooklyn, N. Y., **ALR**
ΩKrebs, Adolph, '13 ..... 500 Penn av., Pittsburgh, Pa., **OP**
Kremen, Abraham, '41 ..... 2355 Eutaw place, Baltimore, Md., **OP**
ΦKress, George H., '18 ..... Apt. 506, 2200 Leavenworth, San Francisco, Calif., **OALR**
Krewson, William Egbert, 3rd, '40 ..... 1930 Chestnut st., Philadelphia, Pa., **OP**
ΩKrieger, George M., '10 ..... 701 Washington, Michigan City, Ind., **OALR**
Krieger, Wm. A., '19 ..... 103 Hooker av., Poughkeepsie, N. Y., **OALR**
Krimsky, Emanuel, '32, Maj. (M.C.), Base Hospital, Army Air Base, Greenville, S. C., **OP**
Krimsky, Joseph, '28 ..... Rio Road, Charlottesville, Va., **OALR**
Krone, William Frank, '42 ..... 31 Lincoln Park, Newark, N. J., **ALR**
Kronenberg, Bernard, '40 ..... 162 W. 54th st., New York, N. Y., **OP**
Kronfeld, Peter C., '31..... 904 W. Adams, Chicago, Ill., **OP**
ΩKrug, Ernest F., '08.... 988 Fifth av., New York, N. Y., **OP**
Kruskal, I. D., '25...101 Lafayette av., Brooklyn, N. Y., **OP**
Krystaf, Joseph G., '37..65 Vernon Place, Buffalo, N. Y., **ALR**
Kuhn, H. S., '30 ..... 112 Rimbach, Hammond, Ind., **OP**
Kuhn, Hugh A., '28 ..... 112 Rimbach, Hammond, Ind., **OALR**
Kully, B. M., '22..3875 Wilshire blvd., Los Angeles, Calif., **ALR**
Kully, Herman E., '35 ..... Medical Arts bldg., Omaha, Nebr., **ALR**
Kulvin, Max M., '31.... 55 E. Washington, Chicago, Ill., **OALR**
Kundert, Fred W., '38 ..... The Monroe Clinic, Monroe, Wis., **ALR**
Kuritzky, Isadore, '40 ..... 1480 Nostrand av., Brooklyn, N. Y., **ALR**

L

Lachman, George S., '37 ..... 450 Sutter, San Francisco, Calif., **OP**
LaClair, Charles Huston, Jr., '40 ..... Maj., 820 W. Marshall, Norristown, Pa., **ALR**
Laff, H. I., '30 ..... Metropolitan bldg., Denver, Colo., **ALR**
ΩLa Force, E. F., '08 ..... Iowa State Bank bldg., Burlington, Iowa, **OALR**
LaGattuta, Frank, '40 .... 3754 White Plains av., Bronx, New York, N. Y., **OP**
Laird, Archibald, '39 ..... Lt. Col. (M.C.), Hamilton Field, Calif., **OP**
Laird, Robert G., '32..Metz bldg., Grand Rapids, Mich., **OP**
Lamb, Harry H., '29 ..... 220 Main, Davenport, Iowa, **ALR**
ΩLamb, R. S., '06..Stoneleigh court, Washington, D. C., **OP**
Lamberson, Frank Abrom, '42 ..... 19600 Grand River, Detroit, Mich., **ALR**
Lamberson, Wm. H., '37 ..... 1st Nat'l Bk. bldg., Colorado Springs, Colo., **ALR**
Lambert, Robert K., '32 ..... 1010 Fifth av., New York, N. Y., **OP**
LaMotte, Wm. O., '35 ..... Medical Arts bldg., Wilmington, Del., **ALR**
Lampert, Max T., '31...Northwest Clinic, Minot, N. D., **ALR**
ΩLancaster, W. B., '13 ..... 520 Commonwealth av., Boston, Mass., **OP**
Landegger, George Paul, '40 ..... 727 W. 7th st., Los Angeles, Calif., **OP**
Landesberg, Jacques, '37 ..... 515 Ocean av., Brooklyn, N. Y., **OP**
Landis, J. E., '30 ..... 232 N. Sixth, Reading, Pa., **ALR**
Landon, H. Robert, '37 ..... Laurelton Professional bldg., Laurelton, N. Y., **ALR**
Lane, Franklin F., '33 ..... Com. (M.C.), U.S.S. Idaho, c/o Postmaster, San Francisco, Calif., **OALR**
Lang, George H., '32 ..... 202 E. Liberty, Savannah, Ga., **ALR**
Lange, Adolph C., '37 ..... 3903 Olive, St. Louis, Mo., **OP**
Lapointe, Jean, '39 ..... 196 Willowdale, Montreal, Can., **ALR**
LaReau, Hector G., '35.. 4803 Lincoln av., Chicago, Ill., **ALR**
ΦΩLarge, S. H., '04 ..... Hanna bldg., Cleveland, Ohio, **ALR**
Larimore, Fred Campbell, '40 ..... 500 Penn av., Pittsburgh, Pa., **OP**
ΣLarkin, Bernard J., '16 ... 23 E. Ohio, Indianapolis, Ind., **OP**
ΣLarsen, Carl L., '16 .. Lowry Med. bldg., St. Paul, Minn., **OALR**
Larson, Bertil T., '38 ..... Riker bldg., Pontiac, Mich., **ALR**
Larson, G. Arthur, '33.... A.O.U.W. bldg., Fargo, N. D., **OALR**
LaRue, C. L., '22 ..... Highland Clinic, Shreveport, La., **OALR**
Lasky, Mortimer A., '39, Maj., Base Hospital A.T.C., Municipal Airport, Memphis, Tenn., **OP**
ΣLassen, Fritz, '15 ..... Pueblo Clinic bldg., Pueblo, Colo., **OALR**
Lassiter, Chester L., '38 ..... Hamilton Nat'l Bk., Chattanooga, Tenn., **ALR**
Last, Murray A., '36...1123 Park av., New York, N. Y., **OP**
Lathbury, Edwin, '42, Capt. (M.C.), Moore General Hospital, Swannanoa, N. C., **ALR**
Lathrop, Frank Damron, '40, Maj. (M.C.), 541 Lincoln Road, Grosse Pointe, Mich., **ALR**
Latimer, Marvin L., '38 ..... 129 Whitney av., New Haven, Conn., **ALR**
ΦLauber, Prof. Dr. Hans, '34 ..... Ul Gertrudy 18ms, Krakav, Generalgouvernment, **OP**
Lauppe, Frederick Andrew, '40 ..... Capt., 29th Field Hospital, Camp Campbell, Ky., **OP**
Laval, Joseph, '31 ..... 136 E. 64th, New York, N. Y., **OP**
ΣLa Vigne, A. A., '18 .. 667 Madison av., New York, N. Y., **OALR**
Law, Frederick M., '37...140 E. 54th, New York, N. Y., **ALR**

Lawrence, Granville A., '13 ..... Medical Arts bldg., Philadelphia, Pa., **OP**
Lawson, Lawrence J., '31 ... 636 Church, Evanston, Ill., **ALR**
Lawwill, Stewart, '28 ..... Medical Arts bldg., Chattanooga, Tenn., **OALR**
Lazar, Abraham M., '38 ..... Maj., Station Hospital, Huntsville Arsenal, Ala., **ALR**
Lazar, Nathan K., '36 ... 58 E. Washington, Chicago, Ill., **OP**
Leach, Robert Swepson, '41 ..... 422 W. Cumberland av., Knoxville, Tenn., **OP**
Leahey, Brendan D., '36 ... 9 Central av., Lowell, Mass., **OP**
Leahey, George A., '37 .... 45 Merrimack, Lowell, Mass., **OP**
Leamer, Bruce V., '38 ..... Patrol Wing No. 8, Naval Air Station, Norfolk, Va., **ALR**
Leasure, J. Kent, '31 ..... Hume-Mansur bldg., Indianapolis, Ind., **ALR**
Leavenworth, R. O., '30 ..... Lowry Med. bldg., St. Paul, Minn., **OP**
Leavy, C. A., '12 .... Metropolitan bldg., St. Louis, Mo., **ALR**
Lebensohn, James E., '27, Lt. Comdr. (M.C.), 20 E. Delaware place, Chicago, Ill., **OALR**
Le Compte, E. D., '27 ..... 9 Exchange pl., Salt Lake City, Utah, **OALR**
Lederer, Francis L., '25 ..... Lt. Comdr., U. S. Naval Hospital, Philadelphia, Pa., **ALR**
Leech, V. M., '30 ..... 55 E. Washington, Chicago, Ill., **OP**
LeFevre, Willis H., '35 ... 234 N. Duke, Lancaster, Pa., **ALR**
Lefkon, Irving M., '39 ..... 37-14 73rd, Jackson Heights, N. Y., **ALR**
Lefler, Anna B., '18 .610 S. Broadway, Los Angeles. Calif., **OALR**
Leggett, Albert E., '34 .... Breslin bldg., Louisville, Ky., **OP**
Lehan, James W., '16 .... 1620 12th av., Greeley, Colo., **OALR**
Lehrfeld, Louis, '39 ..... 1321 Spruce, Philadelphia, Pa., **OP**
Leich, Charles Francis, '41, Lt. (M.C.), U. S. Naval Air Training Center Dispensary, Pensacola, Fla., **ALR**
Leinfelder, Placidus J., '37 ..... University Hosp., Iowa City, Iowa, **OP**
LeJeune, F. E., '27 ... Ochsner Clinic, New Orleans, La., **ALR**
Lemel, Samuel L., '35 .1030 Euclid av., Cleveland, Ohio, **ALR**
Lemere, H. B., '12 ..... 9615 Brighton Way, Beverly Hills, Calif., **OALR**
Lemoine, A. N., '20 .... Rialto bldg., Kansas City, Mo., **OP**
Lennon, Robert Watson, '41 .... Morris bldg., Joliet, Ill., **OP**
Lentine, Joseph, '38, Capt. (M.C.), Headquarters 3rd Air Force, Tampa, Fla., **ALR**
Lepard, Cecil W., '37 ..... David Whitney bldg., Detroit, Mich., **OP**
Lerner, Macy L., '23 .... 332 Park av., Rochester, N. Y., **OP**
Leshin, Norman, '31 .... 5480 Cornell av., Chicago, Ill., **ALR**
Lester, Harry S., '09 ..... Murray bldg., Streator, Ill., **OALR**
Le Tellier, Forrest S., '36 ..... Doctors bldg., Knoxville, Tenn., **OP**
Levbarg, John J., '37 ..... Tilton General Hospital, Fort Dix., N. J., **ALR**
Levin, Abram A., '37 .... Carew Tower, Cincinnati, Ohio, **OP**
Levin, Nathaniel M., '39, Comdr. (M.C.), Naval Air Station, Richmond, Fla., **ALR**
Levitt, Jesse M., '35, Capt. (M.C.), Army Medical Center, Washington, D. C., **OP**
Levy, Abram, '34 ..... 507 Church, Bound Brook, N. J., **OP**
Levy, Harry H., '31 .... 119 Murray, Binghamton, N. Y., **OP**
Levy, Irvin, '39 ..... 154 W. State, Trenton, N. J., **OP**
Levy, Louis, '16 .. Commerce Title bldg., Memphis, Tenn., **ALR**
Levy, Robert, '96 .... Metropolitan bldg., Denver, Colo., **ALR**
Lewin, Michael, '42, Capt. (M.C.), ... Hammond General Hospital, Modesto, Calif., **ALR**
Le Win, Thurber, '29 ... 112 Linwood av., Buffalo, N. Y., **OP**
Lewis, A. C., '16 .... 130 Madison av., Memphis, Tenn., **OALR**
Lewis, Fielding O., '21 ..... (Retired), Media, Pa., **ALR**
Lewis, George A., '20 ..... 213 Main, Roundup, Mont., **OALR**
Lewis, J. D., '21 ..... 724 4th st., Marysville, Calif., **OALR**
Lewis, J. P., '15 .. 479 Commonwealth av., Boston, Mass., **ALR** and 681 Main, Waltham, Mass.,
Lewis, Philip M., '28 ... Exchange bldg., Memphis, Tenn., **OP**
Lewis, Samuel J., '37 ..... S. F. C. bldg., Augusta, Ga., **ALR**
Lewy, Alfred, '42 .... 25 E. Washington st., Chicago, Ill., **ALR**
Lewy, Robert B., '39 .... 25 E. Washington, Chicago, Ill., **ALR**
Leyda, James H., '27 .Metropolitan bldg., Denver, Colo., **ALR**
Lierle, Dean M., '28 ..... University Hospital, Iowa City, Iowa, **ALR**
Lifschutz, Jacob, '25 .. 310 S. Michigan av., Chicago, Ill., **OALR**
Liggett, Harold, '36 ..... 133 E. 58th, New York, N. Y., **ALR**
Light, George, '37 ..... 709 N. 11th, Milwaukee, Wis., **ALR**
Lillie, Harold I., '20 .... Mayo Clinic, Rochester, Minn., **ALR**
Lillie, W. Ivan, '30 ..... 255 S. 17th, Philadelphia, Pa., **OP**
Limbach, Earl Frederick, '40 ..... 30 W. 59th, Station G, New York, N. Y., **ALR**
Lindberg, Vernon Leslie, '42, Capt. (M.C.), Physicians' and Surgeons' bldg., Minneapolis, Minn., **OP**
Lindsay, John R., '36 ..... 950 E. 59th, Chicago, Ill., **ALR**
Lingeman, B. N., '30 ..... Ben Hur bldg., Crawfordsville, Ind., **ALR**
Lingeman, E. L., '29 ..... Hume-Mansur bldg., Indianapolis, Ind., **ALR**
Linn, J. G., '19 ..... Jenkins Arcade, Pittsburgh, Pa., **OP**
Linton, Clair S., '37 .... Maj. (M.C.), 525 N. Wilson av., Tucson, Ariz., **ALR**
Lischkoff, M. A., '13 ..... Blount bldg., Pensacola, Fla., **OALR**
Lister, Sir William T., '23 ..... 24 Devonshire pl., W. 1, London, Eng.,
Litchfield, J. T., '21 ..... Medical Arts bldg., Minneapolis, Minn., **OALR**
Littell, J. Jerome, '31 ..... Hume-Mansur bldg., Indianapolis, Ind., **ALR**
Little, Milton F., '38 ..... 49 Pearl, Hartford, Conn., **OP**
Littwin, Charles, '33 ..... 950 Queen Anne road, Teaneck, N. J., **OALR**
Liva, Arcangelo, '40 .. 5 Pangborn pl., Hackensack, N. J., **OP**
Livingston, George S., '30 ..... 185 N. Wabash av., Chicago, Ill., **ALR**
Livshin, Norman, '36, Lt. Comdr. (M.C.), U. S. Navy, Naval Hospital, Parris Island, S. C., **ALR**
Lloyd, A. W., '15 ..... Lloyd bldg., Hammond, Ind., **OALR**
Lloyd, Ralph I., '25 ..... 14 Eighth av., Brooklyn, N. Y., **OP**
Lockwood, Chester Clinton, '42 ..... Capt. (M.C.), 4707 Broadway, Chicago, Ill., **OP**
Loeb, William J., '42, Capt. (M.C.), 1560 S. U. Medical Section, Station Hospital, Camp Atterbury, Ind., **ALR**
Loewe, Walter R., '34 ..... Comdr. (M.C.), U. S. Navy (address unknown), **OP**
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Logue, Lyle J., '31 ..... 1304 Walker, Houston, Texas, **ALR**
Lokey, H. M., '09 ..... Medical Arts bldg., Atlanta, Ga., **OALR**
Lombardo, Melchiore, '23 ..... 142 Joralemon, Brooklyn, N. Y., **OP**
Long, John C., '36 .. Metropolitan bldg., Denver, Colo., **OP**
Long, Samuel H., '39 ..... Medical Arts bldg., Chattanooga, Tenn., **ALR**
Loomis, E. A., '18 ..... Medical Arts bldg., Minneapolis, Minn., **OALR**
Looper, Edward A., '23 ..... 104 W. Madison, Baltimore, Md., **OALR**

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1930 Wilshire blvd., Los Angeles, Calif., **OP**
Lore, John M., '36 325 West End av., New York, N. Y., **ALR**
Lovesey, Burton E., '39
76 Bay State road, Boston, Mass., **ALR**
Lowenthal, Gerson, '35, Capt. (M.C.),
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Luber, Samuel, '38
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Lubman, Max, '19 50 Manhattan av., New York, N. Y., **ALR**
Lucas, Harold R., '28
Medical-Dental bldg., Portland, Ore., **OALR**
Ludwig, William Henry, '40
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(S)Luedde, W. H., '16 Metropolitan bldg., St. Louis, Mo., **OP**
Luhr, Alfred F., '31 40 W. North, Buffalo, N. Y., **OP**
Luke, William R. F., '38
Medical Arts bldg., Toronto, Can., **OP**
(S)Lukens, Charles, '06 316 Michigan, Toledo, Ohio, **OALR**
Lukens, J. A., '30 316 Michigan, Toledo, Ohio, **ALR**
Lukens, Robert McD., '20
1923 Spruce, Philadelphia, Pa., **ALR**
Lundon, A. E., '27 Col., 1463 Bishop st., Montreal, Can., **ALR**
Lundvick, Cyril V., '38
Medical Arts bldg., Tacoma, Wash., **OP**
Luongo, Romeo A., '31 2054 Locust, Philadelphia, Pa., **ALR**
Lupo, Carl W., '32, Col. (M.C.), 113th Station Hospital,
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Lurie, M. H., '30 483 Beacon, Boston, Mass., **ALR**
Lux, Paul, '25 Bryant bldg., Kansas City, Mo., **ALR**
Lyle, Donald J., '27 Carew Tower, Cincinnati, Ohio, **OP**
Lyman, Harry W., '23 308 N. 6th, St. Louis, Mo., **ALR**
Lynch, Joseph Sidney, '42, Capt. (M.C.),
A.A.F.T.T.C., Truax Field, Madison, Wis., **OP**
Lynch, Mercer G., '39, Capt. (M.C.),
2024 Audubon st., New Orleans, La., **ALR**
Lynch, Robert J., '31 Equitable bldg., Des Moines, Iowa, **ALR**
Lyons, H. R., '22 6 N. Michigan av., Chicago, Ill., **ALR**

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Schwartz, Leo, '35.....136 E. 57th, New York, N. Y., **ALR**

Schwartz, V. J., '29.....
.....Medical Arts bldg., Minneapolis, Minn., **ALR**

Schwartz, Wm. A., '28....15 E. Monroe, Phoenix, Ariz., **OALR**

Sciarretta, Sylvio A., '31. 55 E. Washington, Chicago, Ill., **ALR**

Scobee, J. Edwin, '40, Lt. Comdr. (M.C.),
334 S. Reeves Drive, Beverly Hills, Calif., **ALR**

Searcy, H. B., '12.....2501 Sixth st., Tuscaloosa, Ala., **OALR**

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.....Hume-Mansur bldg., Indianapolis, Ind., **ALR**

Secoy, F. L., '22.....Davidson bldg., Sioux City, Iowa, **OALR**

Secoy, Harry R., '35.....Central bldg., Everett, Wash., **ALR**

Sedgwich, O. W., '17.....Ionia, Mich., **OALR**

Sedwick, W. A., '16.....Republic bldg., Denver, Colo., **OP**

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10 Dryads Green, Northampton, Mass., **ALR**

Segal, Samuel, Jr., '33, Lt. Comdr. (M.C.), U. S. Naval
Base, Cape May, N. J., **ALR**

Seidel, U. D., '34.....447 Belvedere, LaJolla, Calif., **ALR**

Seletz, Abraham, '36.....
.....Medical Arts bldg., Charleston, W. Va., **ALR**

Selfridge, G. L., '08.....
.....1000 California st., San Francisco, Calif., **ALR**

Seligman, Albert E., '39.....
....41 Forest av., Staten Island, New York, N. Y., **ALR**

Seligstein, Milton B., '26.....
.....Union Planters Bank bldg., Memphis, Tenn., **OALR**

Selinger, Elias, '35....104 S. Michigan av., Chicago, Ill., **OALR**

Selinger, Samuel, '33.....
.....413 Sixtieth st., West New York, N. J., **OALR**

Sellers, L. M., '20.....1719 Pacific, Dallas, Tex., **ALR**

Seltz, Samuel, '31.....Doctors bldg., Cincinnati, Ohio, **ALR**

Seltzer, Albert Pincus, '42.....
.....1332 N. Franklin st., Philadelphia, Pa., **ALR**

Semenov, Herman, '35, Maj. (M.C.),
415 N. Camden drive, Beverly Hills, Calif., **ALR**

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Senturia, Ben H., '39.....
..Capt. (M.C.), 4475 W. Pine blvd., St. Louis, Mo., **ALR**

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...430 El Escarpado, Stanford U., Palo Alto, Calif., **ALR**

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.....First Nat'l Bank bldg., Wichita, Kan., **ALR**

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Maj. (M.C.), E. Greenman Road, Haddonfield, N. J., **ALR**

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.....7036 Jenkins Arcade, Pittsburgh, Pa., **OP**

Shaffer, Harry Gard, '40. 225 Cherry st., Clearfield, Pa., **ALR**

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Shannon, Clarence Wm., '31.....
.....New 4th & Pike bldg., Seattle, Wash., **OP**

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Shannon, L. M., '36. 66 S. Fullerton av., Montclair, N. J., **ALR**

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Shapin, Sydney, '37...1346 Fiftieth st., Brooklyn, N. Y., **ALR**

Shapira, Theo. M., '34. 58 E. Washington, Chicago, Ill., **OP**

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Station Hospital, Gulfport Field, Gulfport, Miss., **OALR**

Shastid, T. H., '09.....202 W. Superior, Duluth, Minn., **OP**

Shawhan, Glenn E., '13.....
.....Dewey Scales bldg., Nampa, Idaho, **OALR**

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Sheahan, William L., '26.....
.....59 College, New Haven, Conn., **OALR**

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Station Hospital, Camp Gordon, Augusta, Ga., **ALR**

Shearer, Harry A., '39...405 E. Grand av., Beloit, Wis., **ALR**

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.....Lowry Med. Arts bldg., St. Paul, Minn., **OP**

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Shepherd, Elmer Roberts, '41 ..... 1746 K st. N.W., Washington, D. C., **ALR**

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Sherman, A. Russell, '33 ..... 671 Broad, Newark, N. J., **OP**

Ⓓ Sherman, Elbert S., '12 ..... 671 Broad, Newark, N. J., **OP**

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Ⓢ Shuman, G. H., '17 ..... 351 Fifth av., Pittsburgh, Pa., **OP**

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Shurtleff, Raymond S., '41 ..... Box 786, Lynch, Ky., **ALR**

Shuster, B. H., '29 ..... 1824 Pine, Philadelphia, Pa., **ALR**

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Ⓢ Silbernagel, C. E., '18 ..... 247 E. State, Columbus, Ohio, **OALR**

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Silverberg, Samuel Joshua, '40 ..... 201 Park st., New Haven, Conn., **ALR**

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Simonton, Kinsey Macleod, '41 ..... Mayo Clinic, Rochester, Minn., **ALR**

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Simpson, M. B., '30 ..... Professional bldg., Kansas City, Mo., **ALR**

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Ⓓ Simpson, W. Likely, '11 ..... Physicians & Surgeons bldg., Memphis, Tenn., **OALR**

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Sloane, H. O., '30 ..... 1717 Pine, Philadelphia, Pa., **OP**

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Smith, Henry B., '37 131 Fulton av., Hempstead, N. Y., **ALR**
Smith, Henry C., '38, c/o Alex Stevenson,
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Detachment, Camp Claiborne, La., **OP**
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Smith, James W., '37..1016 Fifth av., New York, N. Y., **OP**
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Station Hospital No. 2, Fort Jackson, S. C., **ALR**
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Smith, Leon, '39.....467 Linwood av., Buffalo, N. Y., **ALR**
Smith, Louis S., '38.....2017 63rd, Brooklyn, N. Y., **ALR**
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.....421 Huguenot, New Rochelle, N. Y., **ALR**
Smith, Robert Arvil, '41, Maj. (M.C.), Box 6063,
Fitzsimons General Hospital, Denver, Colo., **ALR**
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.....224 W. Washington av., Madison, Wis., **ALR**
(L)Smith, Victor C., '12.....
.....American Bank bldg., New Orleans, La., **OP**
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.....314 Wareham bldg., Hagerstown, Md., **ALR**
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.....Medical Arts bldg., Salt Lake City, Utah, **OP**
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Smithies, Harold R., '37.....
.....2241 Central av., Alameda, Calif., **ALR**
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.....130 Washington, Hagerstown, Md., **ALR**
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.....Medical Arts bldg., Grand Rapids, Mich., **ALR**
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.....B.A.A.F., Station Hospital, Blytheville, Ark., **OP**
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.....1100 Park av., New York, N. Y., **ALR**
Snyder, George Gordon, '40.....
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.....324 W. Main, Frankfort, Ky., **ALR**
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.....1608 Milwaukee av., Chicago, Ill., **ALR**
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.....Power & Light bldg., St. Petersburg, Fla., **ALR**
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also 524 North av., New Rochelle, N. Y.,
Soper, Gail R., '33.....636 Church, Evanston, Ill., **OP**
Sowers, Alva B., '37.....
.....6 N. Michigan av., Sec. 2, Chicago, Ill., **OP**
Spaeth, Edmund B., '28.....
.....1930 Chestnut, Philadelphia, Pa., **OP**
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.....Brotherhood bldg., Kansas City, Kan., **ALR**
Spalding, Lansford M., '39.....
.....185 Fourteenth st., Astoria, Ore., **ALR**
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.....4701 Fifteenth av., Brooklyn, N. Y., **OP**
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Sparks, Alan Leo, '40.....
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Speas, William Paul, '41.....
.....Reynolds bldg., Winston-Salem, N. C., **OP**
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.....Medical Arts bldg., Houston, Tex., **OP**
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(S)Spengler, John A., '16.....423 S. Main, Geneva, N. Y., **OP**
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.....Peoples Bank bldg., Akron, Ohio, **ALR**
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.....208 E. Wisconsin, Milwaukee, Wis., **OALR**
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....224 N. Meridian st., No. 301, Indianapolis, Ind., **ALR**
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.....8838 Woodhaven blvd., Woodhaven, N. Y., **ALR**
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.....Medical Arts bldg., Minneapolis, Minn., **OP**
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.....Medical Arts bldg., Toronto, Can., **ALR**
Statti, Louis Wm., '37..Jenkins Arcade, Pittsburgh, Pa., **OP**
Stauffer, F. Leaver, '34.....
.....Medical Arts bldg., Salt Lake City, Utah, **ALR**
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(S)Steim, J. M., '14.....
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Steinbach, Louis, '37.....
.....1882 Grand Concourse, New York, N. Y., **OP**
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.....Western Montana Clinic, Missoula, Mont., **OP**

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.....First Nat'l Bank bldg., Lexington, Ky., **OP**
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Stewart, T. M., '20.....
.....Union Central bldg., Cincinnati, Ohio, **OP**
Stewart, Walter E., '20.Tribune bldg., Terre Haute, Ind., **OALR**
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.....Burns bldg., Colorado Springs, Colo., **OP**
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Stitt, H. L., '27.....Doctors bldg., Cincinnati, Ohio, **ALR**
Stockwell, Eunice LeBaron, '42.....
.....2018 Pine st., Philadelphia, Pa., **OP**
Stoffregen, Charles Henry, '41.....
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Stoll, K. Ludwig, '29.....19 W. 7th, Cincinnati, Ohio, **OP**
Stone, Harry B., '30..Medical Arts bldg., Roanoke, Va., **ALR**
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Lt., U. S. Naval Hospital, Coco Solo, Canal Zone, **ALR**
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Stoutenborough, Wm. A., '27.....
.....21 E. State, Columbus, Ohio, **OALR**
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Straus, Gerhard David, '41, Lt. (M.C.), Chanute Field, Rantoul, Ill., **ALR**
Strauss, J. F., '25....104 S. Michigan av., Chicago, Ill., **ALR**
Street, Alvin Murray, '41..745 5th av., New York, N. Y., **ALR**
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Strickler, Joseph G., '37.200 W. 57th, New York, N. Y., **ALR**
Strikol, Albert J., '36.....
.....Medical Arts bldg., Wilmington, Del., **ALR**
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Stroud, Sanders Key, '40.....
.....Med. Prof. bldg., Corpus Christi, Texas, **OP**
Strout, G. Elmer, '21.....
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Struble, Gilbert Carl, '42, Maj. (M.C.), Billings ...General Hospital, Fort Benjamin Harrison, Ind., **OP**
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Stueber, Paul J., '17.....209 W. North, Lima, Ohio, **OALR**
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Stutsman, Albert C., '37.....
.....539 N. Grand blvd., St. Louis, Mo., **ALR**
Sudranski, Herbert F., '40.....
.....23 East Ohio st., Indianapolis, Ind., **OP**
Suehs, Oliver W., '41....Norwood bldg., Austin, Texas, **ALR**
Sugar, H. Saul, '41, Capt. (M.C.), Barnes General Hospital, Vancouver, Wash., **OP**
Sullivan, Charles T., '37.....
.....277 Alexander, Rochester, N. Y., **OP**
Sullivan, Clifford Patrick, '40, Capt. (M.C.), 304 W. 63rd st., Chicago, Ill., **OP**
Sullivan, Joseph A., '33..170 St. George, Toronto, Can., **ALR**
Sullivan, Robert E., '32.Doctors bldg., Nashville, Tenn., **OP**
Sullivan, Timothy, '42, Capt. (M.C.), 104 15th av., Sterling, Ill., **ALR**
Sulzman, Frank M., '22....1831 Fifth av., Troy, N. Y., **OALR**
Sulzman, John H., '39, Lt. (M.C.), U.S.N.R., 1831 Fifth av., Troy, N. Y., **OP**
Summers, Wm. S., '27.....
.....David Whitney bldg., Detroit, Mich., **OALR**
Sunde, Einar A., '38.....627 Second, Brooklyn, N. Y., **ALR**
Sutherland, J. M., '29.....Fisher bldg., Detroit, Mich., **ALR**
Sutherland, William S., '40.....
.....1930 Chestnut st., Philadelphia, Pa., **OP**
Swab, Charles M., '28.Medical Arts bldg., Omaha, Nebr., **OP**
Swalm, Charles J., '39.....
.....1330 W. Rockland, Philadelphia, Pa., **ALR**
Swan, John R., '39.....23 E. Ohio, Indianapolis, Ind., **ALR**
Swann, Cecil C., '32...Flat Iron bldg., Asheville, N. C., **ALR**
Swanson, Clifford A., '38, Comdr. (M.C.), U.S.S. Iowa, c/o Fleet Postmaster, New York, N. Y., **ALR**
Swetman, C. R. K., '15.....
.....219 S. Mt. Vernon, Prescott, Ariz., **ALR**
Swick, David A., '37.....30 W. 70th, New York, N. Y., **OP**
Swift, Donald E., '39.....
.....Stark General Hospital, Charleston, S. C., **OP**
Swigert, John L., '37, Maj. (M.C.), Station Hospital, Fort Douglas, Utah, **OP**
Swing, Frank U., '35....Doctors bldg., Cincinnati, Ohio, **ALR**
Sykes, Clarence S., '39..2201 Market, Galveston, Texas, **OP**
Sykes, Edwin Meredith, '42.....
.....Nix Professional bldg., San Antonio, Tex., **ALR**
Syracuse, Victor R., '41.....
.....29 Washington sq. W., New York, N. Y., **OP**

T

Tabor, Gilbert S., '37....11 Park Row, Millerton, N. Y., **ALR**
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.....Union Central bldg., Cincinnati, Ohio, **ALR**
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Taquino, George J., '20...Canal bldg., New Orleans, La., **ALR**
Tarun, William, '10....104 W. Madison, Baltimore, Md., **OP**
Tassman, Isaac S., '29.....
.....Medical Arts bldg., Philadelphia, Pa., **OP**
Taterka, Henry D., '37...302 W. 87th, New York, N. Y., **ALR**
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.....111 W. Adams, Jacksonville, Fla., **ALR**
Taylor, Joseph W., '34.....706 Franklin, Tampa, Fla., **OALR**
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Taylor, Shahane R., '39.....
.....Jefferson bldg., Greensboro, N. C., **ALR**
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.....Professional bldg., Kansas City, Mo., **ALR**
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Tegelberg, Julius J., '39, Lt. Comdr. (M.C.), 12 Monterey road, Worcester, Mass., **ALR**

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1801 Eye st. N.W., Washington, D. C., **ALR**
Terrell, Vance, '42
Stephenville Hospital, Stephenville, Tex., **ALR**
Terry, Theodore L., '32
140 Marlborough, Boston, Mass., **OP**
Thaler, Nathan, '40
570 Eastern Parkway, Brooklyn, N. Y., **ALR**
Theis, Franklyn B., '39 209 S. Broadway, Nyack, N. Y., **ALR**
Thel, Henry C., '38 380 Franklin, Aliquippa, Pa., **ALR**
Theobald, Georgiana Dvorak, '29
715 Lake, Oak Park, Ill., **OP**
Theobald, John J., '29 715 Lake, Oak Park, Ill., **ALR**
Theobald, W. H., '30 307 N. Michigan av., Chicago, Ill., **ALR**
Theodore, Frederick H., '40, Capt. (M.C.),
1210 Lenox av., Miami Beach, Fla., **OP**
(L)Thigpen, C. A., '12 401 S. Court, Montgomery, Ala., **OALR**
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(S)Thomas, Francis W., '16 327 E. State, Columbus, Ohio, **OALR**
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Empire Bank bldg., Clarksburg, W. Va., **OP**
Thomas, James Alanson, '41
Southern Michigan Bank bldg., Coldwater, Mich., **ALR**
Thomas, James W., '28 Sharp bldg., Lincoln, Nebr., **OALR**
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437 W. Houston, Sherman, Tex., **OP**
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(L)Thompson, E. H., '07 19 W. 7th, Cincinnati, Ohio, **OALR**
(L)Thompson, G. H., '09 74 North st., Pittsfield, Mass., **OALR**
Thompson, Howard E., '28 1100 Main, Dubuque, Iowa, **OALR**
Thompson, James Parvin, '42
Capt. (M.C.), Mather Field, Calif., **OP**
Thompson, Lester Emmit, '42
Woolworth bldg., Salida, Colo., **ALR**
(S)Thompson, Peter H., '14
308 Commonwealth av., Boston, Mass., **OP**
(L)Thomsen-von Colditz, G., '06
Dun Huntin, Merritt Island, Fla., **OAL**
Thorburn, O. L., '39, Maj. (M.C.),
Station Hospital, Gunnery School, Las Vegas, Nev., **ALR**
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270 Commonwealth av., Boston, Mass., **OP**
Thorpe, Harvey E., '31 Jenkins Arcade, Pittsburgh, Pa., **OP**
Thorson, John A., '36 Roshek bldg., Dubuque, Iowa, **OP**
Thygeson, Phillips, '34, Maj. (M.C.),
971 Eldorado, Clearwater Beach, Fla., **OP**
Tickle, Thomas G., '36 107 E. 63rd, New York, N. Y., **ALR**
Tiedke, Gunther E., '39, Lt. Comdr. (M.C.),
602 Dream's Landing, W. Annapolis, Md., **OP**
Tilderquist, D. L., '25
Medical Arts bldg., Duluth, Minn., **OALR**
Tillotson, R. S., '30
Medical-Dental bldg., Sacramento, Calif., **ALR**
(L)Timberman, Andrew, '03
91 Hamilton Park, Columbus, Ohio, **OALR**
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(L)Tingley, Louisa Paine, '13
416 Marlborough, Boston, Mass., **OP**
Tippin, Ernest E., '34
1st Nat'l Bank bldg., Wichita, Kan., **ALR**
(L)Tivnen, Richard J., '08 30 N. Michigan av., Chicago, Ill., **OALR**
(S)Tobey, George L., Jr., '17
403 Commonwealth av., Boston, Mass., **ALR**
Todd, Grover C., '22 Jenkins Arcade, Pittsburgh, Pa., **ALR**
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Tolan, Thomas L., '33 Wells bldg., Milwaukee, Wis., **ALR**
Tolman, Henry, '39 136 Federal, Salem, Mass., **ALR**
Tomassene, R. A., '23 1144 Market, Wheeling, W. Va., **OALR**
Tomb, E. H., '30, Maj. (M.C.),
46 Lexington, Framingham, Mass., **ALR**
Tomlinson, John Wesley, '42, Lt. Comdr. (M.C.),
900 Chester Pike, Sharon Hill, Pa., **OP**
Tomlinson, Rolland D., '30
445 E. Broad, Westfield, N. J., **ALR**
(L)Toms, S. W. S., '09 120 S. Broadway, Nyack, N. Y., **OALR**
(S)Tooker, C. W., '17 Carleton bldg., St. Louis, Mo., **OP**
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U. S. Naval Hospital, Bethesda, Md., **OP**
Townes, C. Dwight, '32, Maj. (M.C.), Station Hospital,
S.A.A.C.C., San Antonio, Tex., **OP**
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Germantown Prof. bldg., Philadelphia, Pa., **ALR**
Tracewell, George Logan, '41
Commerce bldg., Okmulgee, Okla., **ALR**
Tracht, Robert R., '41 Lowry bldg., St. Paul, Minn., **OP**
Trainor, George McCague, '42, Capt. (M.C.),
195 Wilshire road, Rochester, N. Y., **ALR**
Traugh, George H., '33
309 Cleveland av., Fairmont, W. Va., **ALR**
Tremble, George E., '32
1390 Sherbrooke st. W., Montreal, Can., **ALR**
Treskow, Frank G., '41 411 E. Mason, Milwaukee, Wis., **ALR**
Trexler, Clarence W., '37, Lt. Comdr. (M.C.),
Young bldg., Honolulu, Hawaii, **ALR**
Trible, G. B., '20
1150 Connecticut av. N.W., Washington, D. C., **OALR**
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Tripp, Leroy R., '20 Security bldg., Sioux City, Iowa, **OALR**
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500 West End av., New York, N. Y., **OP**
Trott, Leslie De Nyse, '42
143 North Brand, Glendale, Calif., **ALR**
Trotter, Homer A., '32 40 W. North st., Buffalo, N. Y., **ALR**
Troup, Wilson J., '38 306 Cherry, Green Bay, Wis., **ALR**
Troutman, Erwin W., '40
21 East State st., Columbus, Ohio, **ALR**
Trowbridge, Barnard Cole, '41
Commerce bldg., Kansas City, Mo., **ALR**
Trowbridge, Dwight H., '39
T. W. Patterson bldg., Fresno, Calif., **OP**
Trueman, Robert Heiserman, '42, Capt. (M.C.),
Stark General Hospital, Charleston, S. C., **OP**
Trull, Alfred C., '38
3 Washington Square, Haverhill, Mass., **OP**
Trumbo, James K., '37 526 Third st., Wausau, Wis., **ALR**
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464 9th st., Brooklyn, N. Y., **ALR**
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Wilshire Med. bldg., Los Angeles, Calif., **ALR**
(S)Turner, H. H., '14 Clark bldg., Pittsburgh, Pa., **OP**
(L)Turner, Oliver W., '09 37 Stone st., Augusta, Maine, **OALR**
Turnley, Wm. H., '33 1 Atlantic st., Stamford, Conn., **ALR**
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Tygett, Glenn J., '39
215 N. Sprigg st., Cape Girardeau, Mo., **ALR**
Tyson, John McClure, '40 3 S. Brady st., DuBois, Pa., **ALR**

U

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.....199 Chestnut, Moorestown, N. J., **ALR**
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Station Hospital, Miami Beach, Fla., **OP**
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Usen, Louis, '36... 278 New York av., Brooklyn, N. Y., **ALR**

V

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Station Hospital, Fort Totten, N. Y., **OALR**
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Van Buskirk, Edmund Linford, '41.....
.....308-312 N. 8th, Lafayette, Ind., **OP**
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.....Rijnsburgerweg 6A, Leiden, Holland,
Vandevere, Wm. E., '32.....
.....1st Nat'l Bank bldg., El Paso, Texas, **OALR**
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.....Chamber of Commerce, Indianapolis, Ind., **ALR**
Van Poole, G. McD., '24 Young bldg., Honolulu, Hawaii, **OALR**
Vanzant, Thomas J., '38.....
..... Medical Arts bldg., Houston, Texas, **OP**
Vaughan, Benjamin H., '38.....
.....Adams bldg., Port Arthur, Texas, **ALR**
Vaughn, E. H., '19.....
.....Peoples Nat'l Bank bldg., Tyler, Tex., **OALR**
Veach, Oscar Lloyd, '40.....
..... Whitney Trust bldg., Sheridan, Wyo., **OP**
Ⓓ Veasey, C. A., Sr., '06.....
.....Paulsen Med. & Dental bldg., Spokane, Wash., **OP**
Veasey, C. A., Jr., '27.....
....Paulsen Med. & Dental bldg., Spokane, Wash., **OALR**
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Lawson General Hospital, Atlanta, Ga., **ALR**
Veitch, Ambert H., '37...170 St. George, Toronto, Can., **ALR**
Verhoeff, F. H., '36.....
.....395 Commonwealth av., Boston, Mass., **OP**
Vermeren, John C., '38.....
...Lt. Comdr. (M.C.), 4753 Broadway, Chicago, Ill., **ALR**
Vermillion, James Stephen, '41, Capt. (M.C.),
First National Bank bldg., Welch, W. Va., **ALR**
Victor, Karl N., '39.....
.....Breslin Medical Arts bldg., Louisville, Ky., **ALR**
Vogel, Felix P. S., '36...1018 E. 163rd, New York, N. Y., **ALR**
Vogt, George C., '37....140 Chapin, Binghamton, N. Y., **ALR**
Ⓓ Voigt, C. B., '12.....1702 Broadway, Mattoon, Ill., **OALR**
Voislawsky, Van Rensselaar, '36.....
.....33 E. 68th, New York, N. Y., **ALR**
Volk, Benjamin M., '38.....
.....146 Washington av., Albany, N. Y., **ALR**
Von der Heydt, Robert, '26.....
.....25 E. Washington, Chicago, Ill., **OP**
von Lackum, John K., '37.....
.....Mer. Nat'l Bank bldg., Cedar Rapids, Iowa, **ALR**
von Zelinski, Walter Franz, '41, Col. (M.C.),
Headquarters N.W. Service Command, A.P.O. 702,
c/o Postmaster, Seattle, Wash., **OP**
Voorhees, Earle W., '35.....
.....8 Garfield pl., Poughkeepsie, N. Y., **ALR**
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8 E. Shadowlawn av. N.E., Atlanta, Ga., **OP**
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.....David Whitney bldg., Detroit, Mich., **OALR**
Votaw, Robert E., '37, Maj. (M.C.), Station Hospital,
Majors Army Air Field, Greenville, Tex., **OALR**
Vrooman, Carl O., '40, Maj. (M.C.), Bradley Field
Station Hospital, Windsor Locks, Conn., **OP**

W

Wachsberger, Alfred, '31, Maj. (M.C.),
Army Medical Center, Washington, D. C., **ALR**
Wagener, Henry P., '26 Mayo Clinic, Rochester, Minn., **OP**
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.....Medical Arts bldg., Oklahoma City, Okla., **ALR**
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..De Merschman Gardens, Grand Junction, Colo., **ALR**
Waldeck, E. A., '34.....
.....238 W. Wisconsin av., Milwaukee, Wis., **OALR**
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4000 Girard av., Philadelphia, Pa., **OP**
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Ⓓ Walker, C. E., '18.....1732 High, Denver, Colo., **OP**
Walker, Charles E., Jr., '37.....
..... Maj. (M.C.), S.A.A.C.C., San Antonio, Tex., **OP**
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Walker, George W., '32.....
.....T. W. Patterson bldg., Fresno, Calif., **ALR**
Walker, John R., '32.....
.....T. W. Patterson bldg., Fresno, Calif., **OP**
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.....Home S. & L. bldg., Youngstown, Ohio, **ALR**
Walker, Oliver Frank, '42.....
.....2873 Lewiston road, Niagara Falls, N. Y., **ALR**
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10 E. Calhoun st., Sumter, S. C., **ALR**
Wall, John William, '42, Lt. (M.C.), U.S.N.R.,
N.C.T.C. Dispensary, Davisville, R. I., **ALR**
Wallerstein, Emanuel U., '41.....
.....Professional bldg., Richmond, Va., **ALR**
Wallner, Linden J., '38.....
.....122 S. Michigan av., Chicago, Ill., **ALR**
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.....640 S. Kingshighway blvd., St. Louis, Mo., **ALR**
Walsh, Thomas J., '39...2059 Parrish, Philadelphia, Pa., **ALR**
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Station Hospital, Perrin Field, Sherman, Tex., **ALR**
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Warner, Raymond C., '39.....
..... 324 E. Wisconsin av., Milwaukee, Wis., **OP**
Warner, Robert J., '29... 706 Church, Nashville, Tenn., **OP**
Warren, William C., Jr., '28 Doctors bldg., Atlanta, Ga., **ALR**
Washick, Frank, '43, Capt. (M.C.),
2205 Meridian av., Miami Beach, Fla., **ALR**
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.....Medical Arts bldg., Omaha, Nebr., **ALR**
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McCloskey General Hospital, Temple, Tex., **OP**

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Weaver, Homer V., '32 ..... 625 Cleveland av. N.W., Canton, Ohio, **ALR**
Weaver, T. W. '14 ..... 261 N. Main, Wichita, Kan., **OP**
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Webster, David H., '37 .. 140 E. 54th, New York, N. Y., **OP**
Webster, F. R., '30 .. State Tower bldg., Syracuse, N. Y., **OP**
Wechsler, A. H., '34 ..... 340 New York av., Brooklyn, N. Y., **ALR**
Weeks, John E., '04 ..... 360 Edgecliff rd., Portland, Ore. (Retired), **OP**
Weeth, C. R., '30 ..... 37 Remsen, Brooklyn, N. Y., **ALR**
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Weih, E. P., '20 ..... 217 Fifth av. S., Clinton, Iowa, **OALR**
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Weil, Sidney, '37 ..... 1840 Grand Concourse, New York, N. Y., **OP**
Weinberg, Herman Charles, '42, Lt. (M.C.), U. S. Army Induction Board No. 10, Clarksburg, W. Va., **OP**
Weinman, Edward B., '39, Capt. (M.C.), 2513 Sunset blvd., Steubenville, Ohio, **OP**
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Weinstein, Philip, '39 .. 400 Ingraham bldg., Miami, Fla., **ALR**
Weinstein, Samuel, '37 .. 441 Ocean av., Brooklyn, N. Y., **ALR**
Weiss, Harry, '40, Capt. (M.C.), 140-50 Ash av., Flushing, N. Y., **OP**
Weiss, J. Allan, '32, Lt. Comdr. (M.C.), U. S. Naval Air Station Dispensary, Trinidad, British West Indies, **ALR**
Weisser, Charles William, '42, Capt. (M.C.), Station Hospital, March Field, Riverside, Calif., **OP**
Weisser, E. A., '17 ..... May bldg., Pittsburgh, Pa., **OP**
Weizenblatt, Sprinza, '38 ..... New Medical bldg., Asheville, N. C., **OP**
Welch, Robert S. G., '22 .. 86 State Circle, Annapolis, Md., **OALR**
Weldon, Wm. A., '33 ..... Glasgow, Ky., **OP**
Wells, David W., '08 .. 31 Bay State road, Boston, Mass., **OP**
Welsh, Thomas F., '34 ..... First Nat'l Bank bldg., Salt Lake City, Utah, **OALR**
Wenaas, Elmer J., '38 .. 24 Wick av., Youngstown, Ohio, **OP**
Wencke, Carl G., '19 ..... 1015 Security Tower, Battle Creek, Mich., **ALR**
Wendel, J. S., '23 .. David Whitney bldg., Detroit, Mich., **ALR**
Wenner, W. T., '30 .. St. Mary's bldg., St. Cloud, Minn., **OP**
Wescott, Virgil, '35 .... 30 N. Michigan av., Chicago, Ill., **OP**
Westervelt, H. O., '32 ..... 239 Pipestone, Benton Harbor, Mich., **ALR**
Westlake, Samuel B., '18 ..... Beaumont Med. bldg., St. Louis, Mo., **ALR**
Wetzel, John O., '30 .. Olds Tower bldg., Lansing, Mich., **OP**
Wexler, David, '30 ..... 103 E. 86th, New York, N. Y., **OP**
Weymuller, Ernest A., '36 ..... 135 E. 64th, New York, N. Y., **ALR**
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Whalman, Harold Frederic, '40 ..... 727 W. 7th st., Los Angeles, Calif., **OP**
Wheeler, Maynard C., '35 .. 30 W. 59th, New York, N. Y., **OP**
Wheeler, Merritt W., '23 .. Lowry bldg., St. Paul, Minn., **OALR**
Whelan, G. L., '25 ..... 2100 Walnut, Philadelphia, Pa., **ALR**
Whelan, William F., '34 .. 2004 Walnut, Philadelphia, Pa., **ALR**
Whisnant, A. M., '13 .. Professional bldg., Charlotte, N. C., **OALR**
Whitaker, Joel, '17 ..... Hume-Mansur bldg., Indianapolis, Ind., **OALR**
White, Carl Haper, '42, Lt. Comdr. (M.C.), Naval Aviation Base, Minneapolis, Minn., **ALR**
White, Clarence Hunt, '42 .. Box 257, Henderson, N. C., **ALR**
White, Francis W., '37 .. 471 Park av., New York, N. Y., **ALR**
White, Herbert T., '37 ..... National bldg., Flint, Mich., **OP**
White, James W., '34 .... 15 Park av., New York, N. Y., **OP**
White, Joshua W., '37 .. Medical Arts bldg., Norfolk, Va., **OALR**
White, Vivian P., '22 ..... Tribune Telegram bldg., Salt Lake City, Utah, **ALR**
Whitney, E. L., '30 .. Henry Ford Hosp., Detroit, Mich., **OP**
Whitney, P. T., '36 ..... 390 Main, Worcester, Mass., **OP**
Whitsell, Fay M., '37, Capt. (M.C.), Army and Navy Gen. Hosp., Hot Springs, Ark., **OP**
Whitworth, John Ewing, '41 .. 49 Hammond, Bangor, Me., **ALR**
Wible, LeRoy E., '35, Lt. Comdr. (M.C.), Port Deposit, Bainbridge, Md., **ALR**
Wiener, Alfred, '08 .... 550 Park av., New York, N. Y., **OALR**
Wiener, Meyer, '12 ..... Box 685, 321 Alameda blvd., Coronado, Calif., **OP**
Wickliffe, Thompson F., '31 .. Tweedy bldg., Jasper, Ala., **ALR**
Wieselthier, Maurice L., '42 ..... 1322 Union st., Brooklyn, N. Y., **OP**
Wiggin, William I. '29 ..... 202 Central, Lowell, Mass., **ALR**
Wilber, Inez Edith, '40 ..... 1746 K st. N.W., Washington, D. C., **OP**
Wilbur, Edward P., '03 ..... American Nat'l Bank bldg., Kalamazoo, Mich., **OALR**
Wiley, Jason L., '39 ..... Metcalf bldg., Auburn, N. Y., **OP**
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.....Grand Forks Clinic, Grand Forks, N. D., **ALR**
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.....789 St. Mark's av., Brooklyn, N. Y., **ALR**

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Semenov, Herman. Maj. (M.C.), 415 N. Camden Drive, ALR

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.....Capt. (M.C.), Letterman General Hospital, **OP**
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Procurement, 703 Market st., **OP**
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.....Lt. (M.C.), U.S.S. Solace, c/o Postmaster, **ALR**
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....Lt. Comdr. (M.C.), Unit 390, c/o Postmaster, **ALR**
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Martin, Paul T......235 E. Santa Clara, **ALR**

SAN LEANDRO

Lower, R. A......Lt. Comdr. (M.C.), 670 Haas av., **ALR**

SAN PEDRO

Earl, Harry D......479 Sixth, **ALR**
Snow, Harold L......639 W. 9th, **OP**

SANTA ANA

Dixon, W. E......1617 E. 4th, **OALR**
Holden, Harold M......
....Maj. (M.C.), Station Hospital, Army Air Base, **ALR**
Maxwell, H. C......1718 N. Main, **OALR**

SANTA BARBARA

Donohoe, Ed. C.....Maj. (M.C.), Hoff General Hospital, **ALR**
Hombach, Frank J......1421 State, **OP**
Jean, G. W......7 E. Valerio, **OP**
Johnston, William H......1515 State, **OALR**
Johnston, William Raby.....
.....Lt. (j. g.) (M.C.), U.S.N.R., 1515 State st., **ALR**
Mellinger, William J......323 E. Sola st., **ALR**

SANTA MARIA

Mesirow, Maurice E......117 E. Cook, **OP**

SOUTH PASADENA

Hall, Lawrence H......1520 Oak st., **OALR**

STOCKTON

Saslaw, Lewis Benjamin.....Bank of America bldg., **ALR**

STOCKTON FIELD

Birge, H. L......Capt. (M.C.), A.F.A.T.S., **OP**

VALLEJO

Dexter, Milton A......416 Georgia, **ALR**
Madeley, Howard Randall.....727 Sonoma, **OP**

VISALIA

Keiper, Geo. F......113 N. Church, **ALR**
Sallander, F. W......212 W. Main, **OALR**

WEST LOS ANGELES

Guggenheim, Louis K......1035 Gayley av. ,**ALR**
Hess, Wm. L......1610 Sawtelle blvd., **OALR**

CALIFORNIA — Continued

WHITTIER

Smith, H. A. — 236 N. Greenleaf av., OALR

WILMINGTON

Smith, W. Jewell — Lt. Comdr. (M.C.), 1222 Avalon blvd., ALR

COLORADO

ALAMOSA

Herriman, L. L. — Physicians bldg., OALR

BOULDER

Fischer, V. B. — Mercantile Bank bldg., OALR
Spencer, Frank R. — Physicians bldg., OALR

COLORADO SPRINGS

Brobeck, von Haller — Ferguson bldg., OP
Chapman, Katharine H. — 304 Burns bldg., OP
Chapman, S. Jefferson — Burns bldg., ALR
Kettelkamp, F. O. — Ferguson bldg., ALR
Lamberson, William Henry — First National Bank bldg., ALR
Neeper, Edward R. — Exchange National Bank bldg., OP
Neeper, Edw. R. — Exchange National Bank bldg., OP
Stine, George H. — Burns bldg., OP

DENVER

Bane, William M. — 1612 Tremont pl., OP
Baum, H. L. — Republic bldg., ALR
Beyer, T. E. — 227 Sixteenth, ALR
Brown, Morrow D. — Republic bldg., ALR
Carmody, T. E. — Metropolitan bldg., ALR
Clark, James W. — Maj. (M.C.), 150 S. Clermont, OP
Collins, E. W. — 1578 Humboldt, ALR
Cooper, Kemp G. — Metropolitan bldg., ALR
Crisp, William H. — Metropolitan bldg., OP
Danielson, Ralph W. — Metropolitan bldg., OP
Davis, Leo Lloyd — 1437 Monaco parkway, OP
Fox, C. H. — 695 S. Canosa court, OALR
Gale, M. Jean — Republic bldg., OP
Greene, Laurence W. — Republic bldg., ALR
Gsell, George F. — Lowry Field, OP
Halsted, Fred S. — Metropolitan bldg., OALR
Hickey, Harold L. — Republic bldg., ALR
Laff, H. I. — Metropolitan bldg., ALR
Levy, Robert — Metropolitan bldg., ALR
Leyda, James H. — Metropolitan bldg., ALR
Long, John C. — Metropolitan bldg., OP
McCaw, John A. — Majestic bldg., OP
McKeown, Elmer E. — Republic bldg., OALR
Marcovo, Maurice E. — 1612 Tremont pl., OP
Murphy, Rex L. — Metropolitan bldg., ALR
Ohmart, Walter A. — Republic bldg., OP
O'Rourke, D. H. — Republic bldg., OP
Porter, Whitney C. — 4310 E. 25th av., OP
Reynolds, Edna M. — 227 Sixteenth, OP
Sedwick, W. A. — Republic bldg., OP
Shields, J. M. — Metropolitan bldg. OP
Smith, Robt. A. — Maj. (M.C.), Box 6063, Fitzsimons General Hospital, ALR
Walker, C. E. — 1732 High, OP

GRAND JUNCTION

Cary, G. C. — Canon block, OALR
Rigg, James P. — 227 N. 5th, ALR
Waldapfel, Richard — De Merschman Gardens ALR

GREELEY

Atkinson, Thomas Ernest — Coronado bldg., ALR
Lehan, James W. — 1620 Twelfth av., OALR
Ringle, Charles A. — 912 Ninth av., ALR

LA JUNTA

Dart, M. O. — Maj. (M.C.), Station Hospital, Army Air Base, ALR
Farnsworth, M. A. — Opera House Apt. bldg., OALR

LONGMONT

Hageman, George Ricker — Longmont Hospital, ALR
Sidwell, Clarence Edward — 608 Fourth av., ALR

PUEBLO

Earnest, Clarence E. — Thatcher bldg., ALR
Hopkins, Guy H. — 702 N. Main, OP
Lassen, Fritz — Pueblo Clinic, OALR
Porter, Geo. L. — Capt. (M.C.), Army Air Base, OP
Rusk, Harvey Schenck — Colorado bldg., OALR

SALIDA

Thompson, Lester Emmit — Woolworth bldg., ALR

STERLING

Elliff, Edgar Alonzo — 216 N. 3rd, ALR
Palmer, Frank E. — Box 267, OALR

TRINIDAD

Richie, L. T. — 100 E. Main, OALR

CONNECTICUT

BRIDGEPORT

DeWitt, Edward Nicholas — 881 Lafayette, OP
Garlick, George B. — 144 Golden Hill, ALR
Kaplan, Leon — 881 Lafayette st., OPA
Smith, Dorland — 881 Lafayette, OPA
Turchik, Frank — 1831 Barnum av., ALR

DANBURY

Sarnoff, Jack — Capt. (M.C.), 16 Fifth av., ALR

GREENWICH

Miller, John — 63 North, ALR
Morrisett, L. E. — Stanwick road, ALR
Yost, W. M. — Lake av., OP

HARTFORD

Bausch, Carl P. — 36 Pearl, OP
Connor, Joseph Joyce — 750 Main st., OP
Davis, James Edward — 179 Allyn, ALR
Hogan, W. L. — 750 Main, OALR
Katz, Dewey — 99 Pratt, OP
Little, Milton F. — 49 Pearl, OP
Neidlinger, William J. — 576 Farmington av., ALR
Snelling, P. W. — 179 Allyn, ALR
Whalen, Edward J. — 750 Main, ALR

MIDDLETOWN

Chase, Carl C. — 121 Main, OP

NEW BRITAIN

Schechtman, Charles T. — 81 W. Main, ALR

NEW HAVEN

Canfield, Norton — Lt. Col. (M.C.), 789 Howard av., ALR
Ferguson, R. J. — 59 College, OALR
Flynn, Charles T. — 41 Thumbull, ALR
Latimer, Marvin L. — 129 Whitney av., ALR
MacCready, Paul B. — 442 Temple, ALR
Sheahan, William L. — 59 College, OALR
Silverberg, Samuel Joshua — 201 Park st., ALR
Sperry, Frederick N. — 107 Whitney, ALR
Yudkin, Arthur Meyer — 257 Church, OP

NEW LONDON

Kenyon, Byron B., Lt. Comdr. (M.C.), Naval Dispensary, Submarine Base, ALR
Wilson, Frank E. — 302 State, ALR

CONNECTICUT — Continued

RAWAYTON

Hurd, L. M.....Box F, **ALR**

STAMFORD

Murray, Henry J.....53 South, **ALR**
Turnley, William H.....One Atlantic, **ALR**

TORRINGTON

Hill, E. S.....51 Main, **ALR**

WATERBURY

Good, William Murray....63 Center, **ALR**

WEST HARTFORD

Smith, E. Terry....P. O Box 42, **OP**

WILLIMANTIC

Arnold, Morton....781 Main, **ALR**

WINDSOR LOCKS

Drooker, J. C.....Capt. (M.C.), Bradley Field, **ALR**
Vrooman, Carl O.....Maj. (M.C.), Bradley Field, **OP**

WOODBRIDGE

Clarke, Clemont C.....Amity road, **OP**

DELAWARE

NEWARK

Cutler, Norman L.....R.F.D. No. 2, **OP**

WILMINGTON

LaMotte, William O.....Medical Arts bldg., **ALR**
Mayerberg, Emil R.....Medical Arts bldg., **ALR**
Pierson, William M.....Medical Arts bldg., **ALR**
Strikol, Albert J.....Medical Arts bldg., **ALR**

DISTRICT OF COLUMBIA

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Alexander, Samuel A.....1830 Eye st. N.W., **ALR**
Andrews, A. H., Jr., Lt. (M.C.), Med. Research Sec. Bureau of Aeronautics, Navy Dept., **ALR**
Asbill, David S.....Capt. (M.C.), Walter Reed Hospital, **OP**
Bell, John Gordon....1835 Eye st. N.W., **ALR**
Bockoven, Sterling....1752 Massachusetts av. N.W., **OP**
Bradley, Jeter C.....900 Seventeenth st., N.W., **ALR**
Breeding, Earl G.....1801 Eye st., N.W., **ALR**
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Constantinople, P. S.....1801 Eye st., N.W., **ALR**
Costenbader, Frank D.....1150 Connecticut av., **OP**
Cox, Ronald Atmore....1779 Massachusetts av., N.W., **OP**
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Davies, H. F.....1835 Eye st., N.W., **ALR**
Davis, David....1835 Eye st., N.W., **ALR**
Davis, Wm. Thornwall....927 Seventeenth st., N.W., **OP**
Dessoff, Joseph....1726 Eye st., N.W., **OP**
Dow, Julian N.....Maj. (M.C.), Walter Reed Hospital, **OP**
Downey, Harold R.....Lt. Comdr. (M.C.), 1740 M st., N.W., **OP**
Enlows, Ella M. A.....1816 R st., N.W., **ALR**
Fischer, Aubrey....1835 Eye st., N.W., **ALR**
Flynn, James A.....1511 Rhode Island av., N.W., **ALR**
Gill, G. Bache....1726 Eye st., N.W., **ALR**
Goodman, Edgar L.....1801 Eye st., N.W., **OP**
Gowen, Thos. F., Naval Dispensary, Navy Dept. bldg., 19th and Constitution av., **ALR**
Grosar, James Noah, Jr.....1740 M st., N.W., **OP**
Hantman, Irvin....Farragut Medical bldg., **ALR**
Henning, Carl....1752 Massachusetts av., N.W., **OP**
House, Hugh O.....1835 Eye st., N.W., **ALR**
Johnson, Don....1835 Eye st., N.W., **ALR**
Kearny, Richard A.....2311 Connecticut av., N.W., **ALR**
King, George L., Jr., Lt. Col. (M.C.), General Dispensary 2D201, Pentagon bldg., **OP**
Knowlton, Don S.....Columbia Medical bldg., **ALR**
Lamb, Robert Scott....Stoneleigh court, **OP**
Levitt, Jesse M.....Capt. (M.C.), Army Medical Center, **OP**
Luber, Samuel....1801 Eye st., N.W., **ALR**
Meloy, William C.....Lt. Comdr. (M.C.), 2019 R st., N.W., **ALR**
Mobley, Marion R.....Lt. Col. (M.C.), Walter Reed Hospital, **ALR**
Moffett, Daniel B.....1150 Connecticut av., N.W., **ALR**
Morgan, William A.....1835 Eye st., N.W., **ALR**
Moss, L. Conner....Lt. Comdr. (M.C.), 2223 Observatory pl., N.W., **OP**
Novick, Joel N.....1726 Eye st., N.W., **ALR**
Pendexter, R. Stevens....Farragut Medical bldg., **OP**
Posner, Adolph....General Dispensary, **OP**
Preefer, C. J.....Capt. (M.C.), Army Medical College, **OP**
Riwchun, M. H.....Maj. (M.C.), Walter Reed Hospital, **OP**
Rones, Benjamin....1610 Eye st., N.W., **OP**
Sawyer, Leroy Lee, Jr.....Farragut Medical bldg., **ALR**
Shepherd, Elmer Roberts....1746 K st., N.W., **ALR**
Sheppard, E. A. W.....927 Seventeenth st., N.W., **OP**
Simpson, George V.....1721 Rhode Island av., N.W., **OP**
Tegge, Charles William....1801 Eye st., N.W., **ALR**
Trible, G. B.....1150 Connecticut av., N.W., **OALR**
Vann, Homer King....410 Cedar st., N.W., **ALR**
Wachsberger, Alfred....Maj. (M.C.), Army Medical Center, **ALR**
Wilber, Inez Edith....1746 K st., N.W., **OP**
Wilkinson, Oscar....1408 L st., N.W., **OALR**
Wilkinson, Richard W.....1408 L st., N.W., **ALR**
Zinkhan, A. M.....1801 Eye st., N.W., **ALR**

FLORIDA

BOCA RATON

Rouse, H. K., Jr.....Maj. (M.C.), Station Hospital, Boca Raton Field, **OP**

CAMP BLANDING

Goldsmith, Jacob....Maj. (M.C.), Station Hospital, **OP**
Held, Albert E. Maj. (M.C.), 37 General Hospital Unit, **ALR**

CAMP HAUN

Simons, Max....Maj. (M.C.), Station Hospital, **ALR**

CLEARWATER

Nickle, Millen Alexander....Box 305, **ALR**
Thygeson, Phillips....Maj. (M.C.), 971 Eldorado, **OP**

CLERMONT

Middleton, A. B.....**OALR**

COCOANUT GROVE

Hutchinson, Abbott T.....4086 Roynolds av., **ALR**

FORT LAUDERDALE

Connor, Arthur B.....Sweet bldg., **ALR**

FORT MYERS

Merrick, Charles Gordon....Leon bldg., **ALR**

JACKSONVILLE

Boyd, Charles Wilson....111 West Adams, **OP**
Knauer, William J.....Buckman bldg., **OALR**
O'Dell, John C., Jr.....Medical Arts bldg., **ALR**
Richardson, Shaler....111 W. Adams, **OP**
Taylor, H. Marshall....111 W. Adams **ALR**
Walls, Edward G.....Lt. Comdr. (M.C.), B.O.Q., Naval Air Station, **ALR**
Wilson, Alpheus K.....St. James bldg., **ALR**

FLORIDA — Continued

KEY WEST

Wattles, F. Merrill...Lt. (M.C.), U. S. Naval Hospital, **ALR**

LAKELAND

Firestone, Charles.....c/o General Delivery, **ALR**

LINDALL FIELD

Rubin, N. S......Maj. (M.C.), **OP**

MERRITT ISLAND

Thomsen-von Colditz, G......Dun Huntin, **OALR**

MIAMI

Black, Nelson Miles.....Huntington bldg., **OP**
Brown, Andrew George.....1010 Huntington bldg., **OP**
Chandler, G. E......Huntington bldg., **ALR**
Crossley, E. R......6821 N. W. 4th av., **OALR**
DeBoe, M. P......Huntington bldg., **OALR**
Dunaway, Carl E......Huntington bldg., **ALR**
Hodson, B. F......1216 S.W. 2nd av., **OALR**
Ingersoll, J. M......1700 S. Bayshore lane, **ALR**
Mendel, James H......Ingraham bldg., **ALR**
Palmer, Bascom H......Huntington bldg., **OALR**
Park, M. B......401 Ingraham bldg., **OP**
Weinstein, Philip.....Ingraham bldg., **ALR**

MIAMI BEACH

Campbell, M. D...Army Air Corps, Mantell Plaza Hotel, **OALR**
Cracovaner, A. J., Maj. (M.C.),
Station Hospital, Miami Beach Air Base Command, **ALR**
Denman, Francis E......541 Lincoln road, **ALR**
Hotchkiss, W. T......541 Lincoln road, **ALR**
McLemore, Carl Sloan.....605 Lincoln road, **ALR**
Repass, R. E......605 Lincoln road, **ALR**
Richardson, John Roberts.....
.....Lt. Comdr. (M.C.), 3880 N. Bayshore Drive, **ALR**
Theodore, F. H......Capt. (M.C.), 1210 Lenox av., **OP**
Unsworth, A. C......Maj. (M.C.), Station Hospital, **OP**
Washick, Frank.....Capt. (M.C.), 2205 Meridian av., **ALR**

ORLANDO

Ingram, Hollis Carlyle.....Capt. (M.C.), 842 Laurel, **OP**
Ingram, L. C......307 S. Orange av., **OALR**
Johnston, Hewitt.....108 E. Central, Box 2002, **ALR**
Nance, Willis O......Angebilt Hotel, **OALR**
Shoemaker, Samuel A......30 E. Church, **OALR**

PENSACOLA

Bullwinkel, H. Griffin...Comdr. (M.C.), Bronson Field, **ALR**
Cook, Raymond C......
.....Lt. Comdr. (M.C.), U.S.N.A.S. Dispensary, **OP**
Gilbert, Joseph G., Lt. Comdr. (M.C.), Naval Hospital,
Naval Air Training Center, **ALR**
Heinberg, Charles J......Medical Center, **ALR**
Leich Charles F., Lt. (M.C.), U.S.N.R.,
U. S. Naval Air Station Dispensary, **ALR**
Lischkoff, M. A......Blount bldg., **OALR**
McLane, Jesse Newman.....Blount bldg., **ALR**
Shipley, John LeRoy.....Naval Air Station Dispensary, **OALR**
Wolpaw, B. J......
Lt. Comdr. (M.C.), Naval Air Station Dispensary, **OP**
Womack, David Ray.....
.....Lt. Comdr. (M.C.), U.S. Naval Hospital, **ALR**

RICHMOND

Levin, Nathaniel M......
.....Comdr. (M.C.), U. S. Naval Air Station, **ALR**

ST. AUGUSTINE

Grace, Charles C......145 King st., **ALR**

ST. PETERSBURG

Cardwell, Edgar P......Maj. (M.C.), A.A.F.T.T.C., **ALR**
Heard, Mary K......(Retired) R.F.D. 2, Box 75, **OP**
McNay, Miller Ost.....472 Central av., **ALR**
Nelson, Orville N......Y. M. C. A. bldg., **OALR**
Post, W. L....Maj. (M.C.), 102 52nd st., Bennett Beach, **OALR**
Solomon, Henry D......Power & Light bldg., **ALR**

SANFORD

Williams, R. J......
Lt. Comdr. (M.C.), Naval Air Station Dispensary, **ALR**

SEBRING

Manigan, Thomas P......Maj. (MC,.). Hendricks Field, **ALR**

TALLAHASSEE

Stoner, Cyrus Hubert, Maj. (M.C.),
Station Hospital, Dale Mabry Field, **OP**

TAMPA

Duke, R. R......706 Franklin, **ALR**
Forbes, Sherman B......706 Franklin, **OALR**
Lentine, Joseph, Capt. (M.C.),
Headquarters 3rd Air Force, **ALR**
Lentine, Joseph.....Capt., Headquarters 3rd Air Force, **ALR**
Lowry, B. W......Citizens Bank bldg., **OALR**
Taylor, Joseph W......706 Franklin, **OALR**

WEST PALM BEACH

Miller, Alice R......314 Vallette Way, **OALR**
Sayad, William Y......Harvey bldg., **OALR**
Strong, James C., Jr......
.....Lt. (M.C.), Station Hospital, Morrison Field, **OP**

GEORGIA

ATLANTA

Baird, James M......Medica l Arts bldg., **OP**
Brawner, Leon Edward.....Medical Arts bldg., **ALR**
Brown, Lester A......478 Peachtree st., N.E., **ALR**
Burgess, Taylor S......Medical Arts bldg., **ALR**
Calhoun, F. Phinizy.....478 Peachtree st. N.E., **OP**
Callahan, Alston.....144 Ponce de Leon av., N.E., **OP**
Campbell, W. E., Jr......Medical Arts bldg., **OP**
Clay, G. E......Medical Arts bldg., **OP**
Equen, M. S......144 Ponce de Leon av., N.E., **ALR**
Hallum, Alton V......478 Peachtree st., N.E., **OP**
Howell, Stacy C......478 Peachtree st., N.E., **OP**
Knisley, Alan D., Maj. (M.C.), Aviation Cadet
Examining Board, 321 New Post Office bldg., **ALR**
Lokey, H. M......Medical Arts bldg., **OALR**
McDougall, J. Calhoun.....Medical Arts bldg., **ALR**
McDougall, William L......Doctors' bldg., **ALR**
Martin, William O., Jr......478 Peachtree st., N.E., **OP**
Veirs, Everett R......
.....Maj. (M.C.), Lawson General Hospital, **ALR**
Vorisek, E. A...Maj. (M.C.), 8 E. Shadowlawn av., N.E., **OP**
Warren, William C., Jr......Doctors' bldg., **ALR**
Wright, Edward S......Medical Arts bldg., **ALR**

AUGUSTA

Diess, Robert G., Capt. (M.C.),
Oliver General Hospital, 4th Service Command, **OP**
Lewis, Samuel J......S. F. C. bldg., **ALR**
Shearer, Geo. M......
.....Maj. (M.C.), Station Hospital, Camp Gordon, **ALR**

CAMP STEWART

Brandfield, I. I......Capt. (M.C.), Station Hospital, **ALR**
Irwin, Thomas M......Maj. (M.C.), Station Hospital, **ALR**

GEORGIA — Continued

CAMP TOCCOA

Coleman, Benjamin.....Capt. (M.C.), Station Hospital, **ALR**

FORT BENNING

Boles, William McDonald, Capt. (M.C.), 24th General Hospital, **OP**

MACON

Smith, J. Allen.....Doctors' bldg., **ALR**
Sporn, Hyman, Maj. (M.C.), Station Hospital, Camp Wheeler, **ALR**
Stewart, James V......Capt. (M.C.), Station Hospital, Cochran Field, **ALR**

ROBINS FIELD

Johnson, Gayle T......Capt. (M.C.), Station Hospital, **ALR**

ROME

Smith, George B......Harbin Hospital, **OALR**

SAVANNAH

Collins, Braswell E., Capt. (M.C.), Station Hospital, Army Air Base, Hunter Field, **ALR**
Good, Palmer W......Capt. (M.C.), Hunter Field, **OALR**
Lang, George Hermanne.....202 E. Liberty, **ALR**

HAWAII

HONOLULU

Howarth, Ervin McIntyre.....1154 Bishop, **ALR**
Moffat, Harold Forster.....881 Young st., **OP**
Pinkerton, Forrest Joy.....Young Hotel bldg., **OALR**
Trexler, Clarence W...Lt. Comdr. (M.C.), Young bldg., **ALR**
Van Poole, G. McD......Young bldg., **OALR**

PEARL HARBOR

Harner, Clyde E...Comdr. (M.C.), U. S. Naval Hospital, **OALR**
Horner, W. D......Comdr. (M.C.), U. S. Naval Hospital, Aica Heights, **OP**

IDAHO

FARRAGUT

Greenlee, Max R......Lt. Comdr. (M.C.), U. S. Naval Hospital, **ALR**

IDAHO FALLS

Battles, Newell H......Capt. (M.C.), 311 N. Placer av., **OALR**

NAMPA

Shawhan, Glenn E......Dewey Scales bldg., **OALR**

POCATELLO

Clothier, Joseph.....Kane bldg., **OALR**
Pond, Casper W......Carlson bldg., **ALR**

ILLINOIS

ALTON

Jones, F. W......101 E. Broadway, **OALR**
Middleton, Harry E......206A W. 3rd, **OALR**
Rawlins, Grove A......206A W. 3rd, **ALR**

AURORA

Darmer, George A......Terminal bldg., **OALR**

BELLEVILLE

Otrich, Grover C......Commercial bldg., **ALR**
Schwartz, F. O......210 E. Main, **OP**

BLOOMINGTON

Elliott, J. Norman.....Griesheim bldg., **ALR**
Galley, Watson W......1000 N. Main, **OALR**
Hartenbower, George E......Unity bldg., **ALR**
Watkins, Harold R......Griesheim bldg., **ALR**

BLUE ISLAND

Field, Homer Baker.....Capt. (M.C.), 2433 W. Prairie, **OP**

CENTRALIA

Hirschfelder, Max Kurt.....408 W. 2nd, **OP**

CHAMPAIGN

Albers, Ed. C......104 W. Clark, **OP**
Ford, Hanby Lewis.....Robeson bldg., **OALR**
Youngerman, William M......104 Clark, **ALR**

CHICAGO

Ackerman, Walter G......122 S. Michigan av., **OP**
Allen, Thomas Dyer.....122 S. Michigan av., **OP**
Andrews, Albert H......25 E. Washington, **OALR**
Apple, Carl.....55 E. Washington, **OP**
Barnett, I. F......30 N. Michigan blvd., **OALR**
Becker Oscar J......310 S. Michigan av., **ALR**
Beem, Ione F......30 N. Michigan av., Sec. 2, **OALR**
Bellows, John.....720 N. Michigan, **OP**
Berger, Louis.....30 N. Michigan av., **OP**
Bernheimer, L. Benno.....104 S. Michigan av., **ALR**
Blonder, Edwin J......185 N. Wabash av., **ALR**
Bothman, Louis.....310 S. Michigan av., **OP**
Brandenburg, Nora B......720 N. Michigan av., **ALR**
Brawley, F. E......30 N. Michigan av., **OP**
Brunner, Hans.....1853 Polk, **ALR**
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Coombs, A. J......30 N. Michigan av., **ALR**
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Delph, John F......700 N. Michigan av., **ALR**
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Dougherty, Clifford L......55 E. Washington, **ALR**
Edelman, Maurice I......849 Gunnison, **ALR**
Fabricant, Noah D......185 N. Wabash av., **ALR**
Findlay, E. K......30 N. Michigan av., **OP**
Fisher, W. A......30 N. Michigan av., **OALR**
Fishman, Louis Z......30 N. Michigan av., **ALR**
Flake, George F......25 E. Washington, **OALR**
Fitzgerald, J. P......30 N. Michigan av., **OP**
Fowler, Earl B......55 E. Washington, **OP**
Fox, Noah.....841 E. 63rd, **ALR**
Frank, Ira.....104 S. Michigan av., **ALR**
Friedberg, Stanton A......Capt. (M.C.), 455 Barry av., Lincoln Park Station, **ALR**
Friedman, Elmer Arthur.....55 E. Washington , **ALR**
Gamble, Richard Cotter.....30 N. Michigan av., **OP**
Geiger, Arthur Henry.....554 Fullerton Parkway, **ALR**
Gifford, Sanford R......720 N. Michigan, Suite 1400, **OP**
Glatt, Morris A......55 E. Washington, **ALR**
Goldenburg, Michael.....104 S. Michigan av., **OP**
Gradle, H. S......58 E. Washington, **OP**
Greenwood, Glenn J......55 E. Washington, **ALR**
Guibor, George P......30 N. Michigan av., **OP**
Gustafson, Olga Frideberg.....185 N. Wabash av., **OP**
Guttman, M. Reese.....30 N. Michigan av., **ALR**
Hagens, Elmer William.....30 N. Michigan, **ALR**
Hall, Alice K......25 E. Washington, **ALR**
Hayden, Jerome William.....4458 Madison st., **ALR**
Hill, Howard.....Maj. (M.C.), A.A.F.T.S., 720 S. Michigan blvd., **ALR**

ILLINOIS — Continued

Hill, Howard, '28.....720 S. Michigan blvd., **ALR**
Hipskind, Myron Maxwell.....6060 Drexel av., **ALR**
Hoffman, L. G......30 N. Michigan av., **OALR**
Hoffman, William A......30 N. Michigan, Sec. 2, **OP**
Holinger, Paul H......700 N. Michigan av., **L**
Hollender, Abraham R......307 N. Michigan av., **ALR**
Howard, William H......9152 Commercial av., **OP**
Jacobs, Milton.....122 S. Michigan av., **OP**
Jordan, George T......30 N. Michigan av., **OALR**
Judd, Delbert K......950 E. 59th, **ALR**
Kammerling, Theodore S......616 N. Central av., **OALR**
Kaufman, Samuel I......185 N. Wabash av., **OP**
Kerwin, Raymond W......310 S. Michigan av., **OALR**
Klien, Bertha A......58 E. Washington, Sec. 2, **OP**
Kraft, Oscar H......1518 N. Dearborn Parkway, **OP**
Krause, Arlington C......950 E. 59th, **OP**
Kronfeld, Peter C......904 W. Adams, **OP**
Kulvin, Max M......55 E. Washington, **OALR**
LaReau, Hector G......4803 Lincoln av., **ALR**
Lazar, Nathan K......58 E. Washington, **OP**
Lebensohn, James E......
.....Lt. Comdr. (M.C.), 20 E. Delaware place, **OALR**
Leech, Vernon M......55 E. Washington, **OP**
Leshin, Norman.....5480 Cornell av., **ALR**
Lewy, Alfred.....25 E. Washington st., **ALR**
Lewy, Robert B......In Service, 25 E. Washington, **ALR**
Lifschutz, Jacob.....310 S. Michigan av., **OALR**
Lindsay, John R......950 E. 59th, **ALR**
Livingston, George S......185 N Wabash av., **ALR**
Lockwood, Chester Clinton.....
.....Capt. (M.C.), 4707 Broadway, **OP**
Lockwood, Chester Clinton.....Capt., 4707 Broadway, **OP**
Lyons, H. R......6 N. Michigan av., **ALR**
McBean, George M......25 E. Washington, **OALR**
McBride, Linn Frederick.....122 S. Michigan av., **ALR**
McIntyre, George Fillmore.....25 E. Washington, **ALR**
McRoberts, Earl S......55 E. Washington, **ALR**
Mann, William A......30 N. Michigan av., **OP**
Meyer, Samuel J......58 E. Washington, **OP**
Moncreiff, William F......58 E. Washington, **OP**
Morwitz, Samuel M......55 E. Washington, **ALR**
Mundt, G. Henry.....30 N. Michigan av., **OALR**
Muskat, Irving.....25 E. Washington, **OALR**
Nash, Herbert T......31 N. State, **ALR**
Nethercut, Glenway W......25 E. Washington, **OP**
Noonan, W. J......4753 Broadway, **OALR**
Novak, Frank J., Jr......30 N. Michigan av., **ALR**
Nugent, Oscar B......231 W. Washington, **OP**
O'Connor, Philip D......30 N. Michigan av., **OP**
O'Connor, Thomas Philip.....30 N. Michigan av., **ALR**
Olkon, Dora Berkman.....58 E. Washington, **OP**
Orcutt, D. C......55 E. Washington, **OALR**
Pearlman, S. J......55 E. Washington, **ALR**
Pellettiere, Edmund Victor.....
.....3414 W. Adams st., Garfield Park Station, **ALR**
Perlman, Henry B......950 E. 59th, **ALR**
Perritt, Richard A......30 N. Michigan av., **OP**
Piszkiewicz, Frank J...Maj. (M.C.), 6019 N. Menard av., **ALR**
Pollack, Samuel.....Capt. (M.C.), 2909 Estes av., **ALR**
Puntenney, Irving.....720 N. Michigan av., **OP**
Roling, Edward A......2753 W. North av., **OP**
Rose, Percy J......104 S. Michigan av., **ALR**
Salinger, Samuel.....25 E. Washington, **ALR**
Savitt, Louis.....3403 Lawrence av., **ALR**
Schaub, Carl Francis.....31 N. State, **OP**
Scheribel, Karl J......55 E. Washington, **OP**
Sciaretta, S. A......55 E. Washington, **ALR**
Selinger, Elias.....104 S. Michigan av., **OALR**
Shambaugh, George E., Jr......122 S. Michigan av., **ALR**
Shapira, Theodore M......58 E. Washington, **OP**
Shapiro, S. L......185 N. Wabash av., **ALR**
Smiley, William A......104 S. Michigan av., **ALR**
Smith, Hiram J......30 N. Michigan av., **OP**
Snitman, Maurice F......55 E. Washington st., **ALR**
Soboroff, Simon H......1608 Milwaukee av., **ALR**
Sowers, Alva Boyd.....6 N. Michigan av., Sec. 2, **OP**
Steffenson, Olaf.....25 E. Washington, **ALR**
Strauss, J. F......104 S. Michigan av., **ALR**
Sullivan, Clifford Patrick....Capt. (M.C.), 304 W. 63rd, **OP**
Theobald, W. H......307 N. Michigan av., **ALR**
Tindal, John M......5517 W. Fulton, **OALR**
Tivnen, Richard J......30 N. Michigan av., **OALR**
Van Alyea, O. E......135 S. La Salle, **ALR**
Vermeren, John C...Lt. Comdr. (M.C.), 4753 Broadway, **ALR**
Von der Heydt, Robert.....25 E. Washington, **OP**
Wagner, Carl B......30 N. Michigan av., **OALR**
Wallner, Linden J......122 S. Michigan av., **ALR**
Wescott, Virgil.....30 N. Michigan av., **OP**
Woodrow, Jack H......Lt. (M.C.), 501 Surf st., **ALR**
Zekman, Theodore N......58 E. Washington, **OP**

CHICAGO HEIGHTS

Gorrell, T. J. H......16th and Vincennes av., **OALR**

DANVILLE

Baldwin, H. E......139 N. Vermillion, **OALR**
Baldwin, Stephen Glidden.....Adams bldg., **ALR**
Fink, Otto E......9 W. Madison, **OP**

DECATUR

Hubble, William Fields.....Citizens bldg., **ALR**

DIXON

McNichols, W. A......101 First, **OALR**

EAST ST. LOUIS

Hanson, William Ludwig.....Spivey bldg., **ALR**
Spitze, E. C......Murphy bldg., **OP**

ELGIN

Russell, Roland D......Room 808 Professional bldg., **ALR**

EVANSTON

Dodd, Oscar.....636 Church st., **OP**
Droegemueller, Wm. H...Maj. (M.C.), 116 Burnham pl., **OP**
Eubank, Miriam D......944 Maple av., **OP**
Galloway, T. C......636 Church st., **ALR**
Lawson, L. J......636 Church st., **ALR**
Soper, Gail Raney.....636 Church st., **OP**
Williams, Thomas J......1604 Chicago av., **ALR**

FREEPORT

Clark, J. Sheldon.....509 State Bank bldg., **OALR**

GREAT LAKES

DeLozier, Leonard C...Lt. (M.C.), U. S. Naval Hospital, **ALR**
Green, Hyman S......
.....Lt. Comdr. (M.C.), Naval Training Station, **OALR**
Hedlund, G. O., Lt. Comdr. (M.C.), U.S.N.R.,
U. S. Naval Training Station, **ALR**
Richter, Harry Joseph.....U. S. Naval Hospital, **ALR**
Hornbogen, D. P......
.....Lt. Comdr. (M.C.), U. S. Naval Hospital, **OP**
Koebbe, E. E...Lt. Comdr. (M.C.), U. S. Naval Hospital, **ALR**
Richter, H. J......U. S. Naval Hospital, **ALR**

HINSDALE

Hara, H. James.....Hinsdale Sanitarium and Hospital, **ALR**

ILLINOIS — Continued

HOMEWOOD
Dillon, Elmo E. ..... 1954 Ridge road, OALR

JACKSONVILLE
Adams, A. L. ..... Professional bldg., OALR

JOLIET
Lennon, Robert Watson ..... Morris bldg., OP
Woodruff, George H. ..... 250 N. Ottawa, ALR
Woodruff, H. W. ..... 250 N. Ottawa, OALR

KANKAKEE
Geiger, C. W. ..... Arcade bldg., OALR
Gernon, William ..... Capt. (M.C.), 241 S. Chicago av., OP

LA GRANGE
Ehrmann, Evelina W. ..... 327 S. La Grange road, OP

LA SALLE
Woods, R. H. ..... 153 Marquette, OP

LAWRENCEVILLE
Gruppe, K. W. ..... Maj. (M.C.), Army Flying School, ALR

MACOMB
Duntley, G. S. ..... Box 47, OALR

MATTOON
Jones, Florentine B. ..... 416 South 17th, ALR
Voigt, C. B. ..... 1702 Broadway, OALR

MAYWOOD
Spiesman, Irwin G. ..... 1908 St. Charles road, ALR

MOLINE
Flatley, T. J. ..... 1518 Fifth av., OALR

OAK PARK
Bredlau, Ernest A. ..... 119 Le Moyne, ALR
Palmer, Mahlon Phelps ..... 715 Lake, ALR
Theobald, Georgiana Dvorak ..... 715 Lake, ALR
Theobald, John J. ..... 715 Lake, ALR

OLNEY
Fisher, H. Noland ..... Olney Sanatorium Clinic, OP

PARK RIDGE
Riser, Roy O. ..... 120 Main, OP

PEKIN
Glasford, S. T. ..... 416 Elizabeth, OALR

PEORIA
Blickenstaff, A. J. ..... Alliance Life bldg., OALR
Brobst, C. H. ..... 107 High, OALR
McGrath, Philip R. ..... Lt. Comdr. (M.C.), 210 Missouri, ALR
Williams, W. C. ..... Jefferson bldg., OALR

QUINCY
Gabriel, C. K. ..... Illinois State Bank bldg., OALR
Gilfillan, Homer John, Jr. ..... 1416 Main, ALR
Stevenson, Walter ..... W. C. U. bldg., OALR

RANTOUL
Straus, G. D. ..... Lt. (M.C.), Chanute Field, ALR

ROCKFORD
Blomberg, T. E. ..... 501 Seventh, ALR
Fringer, W. R. ..... 109 Sheridan st., OP
Potter, J. J. ..... Gas-Electric bldg., ALR

SCOTT FIELD
Allen, James H. ..... Capt. (M.C.), Station Hospital, OP

SPRINGFIELD
Broadwell, Stuart, Jr. ..... 103½ N. 5th, ALR
Deal, John Francis ..... 522 E. Capitol av., OALR
Kerst, John Arthur ..... Capt. (M.C.), R. R. No. 4, OP

STERLING
Brodrick, Frank W. ..... 302 First av., OALR
Sullivan, Timothy ..... Capt. (M.C.), 104 15th av., ALR

STREATOR
Lester, Harry S. ..... Murray bldg., OALR

TAYLORVILLE
Duncan, Perry E. ..... 101½ E. Main, OP

WINNETKA
Ballenger, Howard C. ..... 723 Elm, ALR

INDIANA

BLOOMINGTON
Geiger, Dillon ..... Capt. (M.C.), Fee Lane, ALR

BLUFFTON
Smith, H. Brooks ..... 303 S. Main, OP

CAMP ATTERBURY
Loeb, Wm. J., Capt. (M.C.), 1560 S. U.
Medical Section, Station Hospital, ALR

CRAWFORDSVILLE
Lingeman, B. N. ..... Ben Hur bldg., ALR

EVANSVILLE
Ravdin, B. D. ..... Hulman bldg., OALR
Ravdin, M. ..... Hulman bldg., OALR
Stanton, Harmon LaM. ..... Hulman bldg., OALR

FORT BENJAMIN HARRISON
Struble, Gilbert Carl
..... Maj. (M.C.), Billings General Hospital, OP

FORT WAYNE
Bulson, Eugene Loring ..... 347 W. Berry, OALR
Gessler, William F. ..... 2902 Fairfield av., OALR

GARY
Carmody, Raymond Francis ..... 504 Broadway, OP

HAMMOND
Kuhn, Hedwig S. ..... 112 Rimbach, OP
Kuhn, Hugh A. ..... 112 Rimbach, OALR
Lloyd, A. W. ..... Lloyd bldg., OALR

HARTFORD CITY
Owsley, Guy A. ..... Lt. Col. (M.C.), 214 N. High, ALR

INDIANAPOLIS
Adams, D. S. ..... Hume-Mansur bldg., OALR
Alvis, Edmond Ochs ..... 23 E. Ohio, OP
Bartley, Donald A. ..... Hume-Mansur bldg., OP
Berry D. F. ..... Bankers Trust bldg., ALR
Brown, David Edward ..... 23 E. Ohio, ALR
Chappell, R. S. ..... Hume-Mansur bldg., ALR
Clark, Cecil P. ..... 23 E. Ohio, OP
Clevenger, W. F. ..... Hume-Mansur bldg., ALR
Craft, Kenneth L. ..... Hume-Mansur bldg., ALR
Ellis, Bert E. ..... Hume-Mansur bldg., ALR
Garrett, John D. ..... Bar Association bldg., ALR
Harding, Myron S. ..... Hume-Mansur bldg., OP
Harris, Carl B. ..... Hume-Mansur bldg., OP
Hughes, W. F. ..... Hume-Mansur bldg., OP
Jones, David E. ..... Chamber of Commerce, ALR
Larkin, Bernard J. ..... 23 E. Ohio, OPR
Leasure, J. Kent ..... Hume-Mansur bldg., AL
Lingeman, Edward Laurel ..... Hume-Mansur bldg., ALR
Littell, J. Jerome ..... Hume-Mansur bldg., ALR
McCaskey, C. H. ..... Guaranty bldg., ALR
Manion, Marlow W. ..... Hume-Mansur bldg., ALR
Masters, Robert J. ..... 23 E. Ohio, OP

ILLINOIS — Continued

Molt, William F. ..... Bankers Trust bldg., ALR
Overman, F. V. ..... Hume-Mansur bldg., ALR
Pfafflin, C. A. ..... Bankers Trust bldg., OALR
Pohl, Robert L., Maj. (M.C.), Headquarters, I Troop Carrier Command, Stout Field, ALR
Row, D. Hamilton ..... Lt. Comdr. (M.C.), 23 E. Ohio, OP
Rutherford, C. W. ..... Hume-Mansur bldg., OP
Sage, Russell A. ..... 23 E. Ohio, ALR
Seaton, Guy W. ..... Hume-Mansur bldg., ALR
Sparks, Alan Leo ..... Maj., 4310 Central av., ALR
Sputh, Carl B. ..... 224 N. Meridian st., No. 301, ALR
Sudranski, Herbert F ..... 23 E. Ohio, OP
Swan, John R. ..... 23 E. Ohio, ALR
Van Osdol, Harry A. ..... Chamber of Commerce bldg., ALR
Whitaker, Joel ..... Hume-Mansur bldg., OALR
Wright, J. William ..... Hume-Mansur bldg., ALR

KOKOMO
Adams, Charles Joseph ..... Armstrong-Landon bldg., OALR

LAFAYETTE
Peterson, Joel A. ..... 609 Lafayette Life bldg., ALR
Van Buskirk, Edmund Linford ..... 308-312 N. 8th, OP

LOGANSPORT
Egan, Burton Wright ..... 2305 Broadway, OALR

MARION
Braunlin, William H. ..... Marion National Bank bldg., OALR

MICHIGAN CITY
Krieger, G. M. ..... 701 Washington, OALR

RICHMOND
Holland, Emory E. ..... 51 S. 8th, OP

SOUTH BEND
Cassady, J. Vernal ..... Sherland bldg., OALR
Dietl, Ernest Lawrence ..... In Service, 1037 Hudson av., ALR
Rigley, Edward L. ..... 107 E. Jefferson blvd., ALR
Savery, C. E. ..... Sherland bldg., OALR

TERRE HAUTE
Allen, Orris T. ..... Rose Dispensary bldg., OALR
Gillum, J. R. ..... 221 S. 6th, OALR
McBride, Noel S. ..... 12 S. 7th, OP
Mahoney, Charles Leo ..... 221 S. 6th, ALR
Stewart, Walter E. ..... Tribune bldg., OALR

UNION CITY
Ruby, F. McK. ..... 235 W. Pearl, OALR

IOWA

BURLINGTON
La Force, E. F. ..... Iowa State Bank bldg., OALR

CARROLL
McConkie, Willis L. ..... ALR

CEDAR RAPIDS
Foster, Wayne J. ..... Higley bldg., ALR
Ivins, H. M. ..... Merchants Nat'l Bank bldg., OALR
von Lackum, John Kenneth ..... Merchants Nat'l Bank bldg., ALR

CLINTON
Blackmar, Francis B. ..... Maj. (M.C.), Schick General Hospital, ALR
Foster, Warren H. ..... 217 Fifth av. S., ALR
Welh, E. P. ..... 217 Fifth av. S., OALR

COUNCIL BLUFFS
Dean, F. W. ..... 22 S. Sixth, OALR
Howard, Lloyd G. ..... Park bldg., OALR
Maiden, Sydner D. ..... Bennett bldg., OALR

CRESTON
Childs, H. A. ..... 1016 W. Adams, OALR

DAVENPORT
Hands, Sidney G. ..... 201 W. 2nd, ALR
Harkness, Gordon F. ..... Putnam bldg., OALR
Hofmann, W. P. ..... Kahl bldg., OALR
Lamb, Harry Howe ..... 220 Main, ALR
Rock, John Emmett ..... Putnam bldg., ALR

DES MOINES
Downing, James A. ..... Bankers Trust bldg., ALR
Jones, Cecil C. ..... Equitable bldg., OALR
Lynch, Robert J. ..... Equitable bldg., ALR
Matheson, John H. ..... Equitable bldg., OP
Pearson, William W. ..... Bankers Trust bldg., OALR

DUBUQUE
Connelly, Edgar ..... 7 Roshek bldg., ALR
Quinn, Francis P. ..... 887 Mt. Carmel road, ALR
Thompson, Howard E. ..... 1110 Main, OALR
Thorson, John A. ..... Roshek bldg., OP

FORT DODGE
Chase, S. B. ..... Carver bldg., OALR
Kluever, Herman C. ..... Lt. Comdr. (M.C.), Carver bldg., OP

FORT MADISON
Reimers, R. S. ..... 613 Eighth, ALR

IOWA CITY
Albright, George C. ..... 104 S. Clinton, ALR
Evers, Lorance Bixler ..... University Hospital, ALR
Leinfelder, Placidus J. ..... University Hospital, OP
Lierle, Dean M. ..... University Hospital, ALR
O'Brien, C. S. ..... University Hospital, OP

LE MARS
Joynt, M. J. ..... Royal bldg., ALR

MARSHALLTOWN
French, R. F. ..... Masonic Temple, OALR
Wolfe, Otis R. ..... Masonic Temple, OP

MASON CITY
Chenoweth, Charlie Everette ..... Forester's bldg., ALR
Fallows, Howard D. ..... Foresters bldg., OALR
O'Brien, Steve A. ..... First Nat'l Bank bldg., OALR

OTTUMWA
Prewitt, Leland Howard ..... 211 E. 2nd, ALR

SHENANDOAH
Maloy, Wayland H. ..... 608½ W. Sheridan av., OP

SIOUX CITY
Decker, J. C. ..... Frances bldg., OALR
Dvorak, Joseph E. ..... Davidson bldg., OP
Gittins, T. R. ..... Davidson bldg., OALR
Reeder, J. E. ..... Davidson bldg., OALR
Secoy, F. L. ..... Davidson bldg., OALR
Tripp, Leroy R. ..... Security bldg., OALR

WATERLOO
Bender, H. A. ..... Black bldg., ALR
Joynt, A. J. ..... 525 Sycamore, OALR
Reuling, F. H. ..... Black bldg., OALR
Russell, R. E. ..... 606 Commercial, OALR

WAVERLY
Robinson, R. E. ..... 205½ E. Bremer av., OALR

KANSAS

ATCHISON
Bribach, Eugene J. ..... Professional bldg., OALR

COFFEYVILLE
Chadwick, Ira B. ..... 717½ Walnut, OALR

CONCORDIA
Robertson, Edward N. ..... New Caldwell bldg., OALR

EMPORIA
Granger, W. B. ..... Gazette bldg., OALR
Trimble, Clarence S. ..... Gazette bldg., OALR

FORT LEAVENWORTH
Rosenbaum, Harry D. ..... Maj. (M.C.), 211 Auger av., OP

FORT RILEY
Schlanser, A. E. ..... Maj. (M.C.), 101 Schofield Circle, OALR

HUTCHINSON
Greever, Boyd L. ..... 100 N. Main, OALR
Quiring, Walter O. ..... 100 First West, ALR

KANSAS CITY
Barry, William B. ..... University of Kansas Hospitals, ALR
Billingsley, John A. ..... Huron bldg., OP
Spake, LaVerne B. ..... Brotherhood bldg., ALR

MANHATTAN
Dyer, B. G. ..... 109½ S. 4th, ALR

NEWTON
Morgan, Harold Eugene ..... 210 S. Pine st., OP

PARSONS
Blasdel, Tyler David ..... 1101½ S. 18th, ALR

PITTSBURG
Haas, Louis Richard ..... Smith Clinic, OALR

PRATT
Pitman, Will D. ..... First Nat'l Bank bldg., ALR

SALINA
Cheney, Ralph E. ..... United Life bldg., OP

TOPEKA
Allen, George V. ..... 835 Kansas av., OALR
Ashley, Byron John ..... 901 Kansas av., ALR
Boggs, Frank C. ..... Mills bldg., OP
Kirkpatrick, Hazen Leon ..... 1320 Pembroke Lane, ALR
Reed, William W. ..... Mills bldg., ALR

WICHITA
Brownell, Morton Emmons ..... 105 N. Main, OP
Carter, E. D. ..... Central bldg., ALR
Cheney, James W. ..... Schweiter bldg., OALR
Gillett, Wilbur G. ..... 201½ N. Main, OP
Gsell, J. F. ..... Beacon bldg , OALR
Maggard, D. I. ..... Orpheum bldg., OALR
Marshall, Hal E. ..... 227 E. Douglas av., ALR
Reifsneider, J. Stanley ..... Beacon bldg., ALR
Seydell, E. M. ..... First Nat'l Bank bldg., ALR
Tippin, Ernest E. ..... First Nat'l Bank bldg., ALR
Weaver, T. W. ..... 201 N. Main, OP

WINFIELD
Ralls, C. T. ..... State Bank bldg., OALR

KENTUCKY

BOWMAN FIELD
Adams, Eldridge S. ..... Col. (M.C.), Station Hospital, ALR

CAMP BRECKENRIDGE
Bornstein, Max. . Maj. (M.C.), Post Hospital, 1570 S.U., ALR

CAMP CAMPBELL
Barone, M. H. ..... Capt. (M.C.), 27th Field Hospital, ALR
Cleary, James A. ..... Maj. (M.C.), 1580th Service Unit, ALR
Lauppe, F. A. ..... Capt. (M.C.), 29th Field Hospital, OP

COVINGTON
Bledsoe, R. W. ..... First Nat'l Bank bldg., OALR

FORT KNOX
Anderson, Geo. H. ..... Capt. (M.C.), Station Hospital, ALR

FRANKFORT
Snyder, William S., Jr. ..... 324 W. Main, ALR

GLASGOW
Weldon, William A. ..... OP

HARLAN
Gunn, Milus Lydell ..... Metcalf bldg., ALR

LEXINGTON
Armstrong, R. M. ..... First National Bank bldg., OALR
DeWeese, Clarence ..... 201 W. Second st., OALR
Marks, Samuel Blackburn ..... 200 W. Second, ALR
Stern, Milton J. ..... First National Bank bldg., OP

LOUISVILLE
Baker, Melvin C. ..... Heyburn bldg., OP
Bass, A. L. ..... Heyburn bldg., OALR
Dean, Walter ..... Francis bldg., OALR
Dulaney, Octavius ..... Brown bldg., ALR
Hall, G. C. ..... Brown bldg., OALR
Heitger, J. D. ..... Heyburn bldg., OALR
Hutcherson, John Kenneth ..... Francis bldg., OP
Juers, Arthur L. ..... Heyburn bldg., ALR
Leggett, Albert E. ..... Breslin bldg., OP
Pfingst, A. O. ..... 1009 Cherokee road, OP
Victor, Karl N. ..... Breslin Medical Arts bldg., ALR

LYNCH
Shurtleff, Raymond S. ..... Box 786, ALR

MADISONVILLE
Garnier, William Hampton ..... Hospital bldg., ALR
Salmon, David Lee ..... Maj. (M.C.), Doctors bldg., ALR

PADUCAH
Abell, Harry Davis ..... 333 Broadway, OP
Pace, Errett ..... City Savings Bank bldg., ALR
Reynolds, H. G. ..... 717 Citizens Savings Bank bldg., OALR

LOUISIANA

ALEXANDRIA
Simmonds, Noel T. ..... 1404 Murray, OP

BATON ROUGE
Jackson, Rufus ..... Louisiana Nat'l Bank bldg., ALR
McLaurin, James Walter ..... 251 3rd, ALR

BARKSDALE FIELD
Ridall, Earle G. ..... Capt. (M.C.), Station Hospital, ALR

CAMP CLAIBORNE
Smith, Herschel S. . Capt. (M.C.), Medical Detachment, OP

LOUISIANA — Continued

CAMP LIVINGSTON

Burke, B. R......Maj. (M.C.), 43rd General Hospital, **ALR**
Marks, Paul Lombroso..Capt. (M.C.), Station Hospital, **OP**
Paulk, James R......Lt. (M.C.), Station Hospital, **OP**
Pelz, Mert D......Maj. (M.C.), Station Hospital, **ALR**

LAKE CHARLES

Moss, Olin W......Weber bldg., **ALR**

NEW ORLEANS

Alexander, Lucian W......Maison Blanche bldg., **ALR**
Bahn, Charles A......Pere Marquette bldg., **OP**
Benz, Alvin H., Capt. (M.C.),
Station Hospital N.O.P.E., Jefferson Branch, **OP**
Blum, Henry N......American Bank bldg., **OP**
Bradish, Robert Francis, Maj. (M.C.), New Orleans
Port of Embarkation, Poland and Dauphin sts., **OP**
Brewster, H. F......Union Indemnity bldg., **ALR**
Brown, Charles L......Union bldg., **ALR**
Buffington, Wiley R......Hibernia Bank bldg., **OP**
Clark, William B......American Bank bldg., **OP**
Corte Passi, Vital E., Capt. (M.C.),
210 General Hospital, A.P.O. 837, c/o Postmaster, **ALR**
Cox, Charles L......Pere Marquette bldg., **ALR**
Crebbin, Alexander R......Maison Blanche bldg., **OP**
Fuchs, Valentine Henry.....American Bank bldg., **ALR**
Goldberg, Sol.....Maj. (M.C.), La Garde Hospital, **OP**
Gooch, John B......Canal Bank bldg., **ALR**
Gronberry, Carl.....Maison Blanche bldg., **ALR**
Irwin, John Joseph.....Baronne bldg., **ALR**
Hanley, Jeanne C. Roeling.....Maison Blanche bldg., **ALR**
Kearney, Harold Leslie.....1403 Delachaise, **ALR**
LeJeune, F. E......Ochsner Clinic, **ALR**
Lynch, Mercer G......Capt. (M.C.), 2024 Audubon st., **ALR**
McComiskey, Arthur James.....3420 Prytania, **ALR**
Mueller, Lawrence Wm......
.....Capt. (M.C.), LaGarde General Hospital, **ALR**
Royals, Walter C......La Garde General Hospital, **OP**
Rubin, M. R., Capt. (M.C.), A.P.O. 832, c/o Postmaster, **ALR**
Smith, Victor C......American Bank bldg., **OP**
Taquino, George J......Canal bldg., **ALR**
Wagner, W. A......200 Carondelet, **ALR**
Weil, A. I......3439 Prytania, **ALR**
Wood, Spencer.....500 Verret, **ALR**

PINEVILLE

Ewing, Fayette C......(Retired), "Nosegay,"

SHREVEPORT

LaRue, C. L......Highland Clinic, **OALR**
Scales, J. L......Medical Arts bldg., **OALR**
Wilkinson, Joel A......1130Louisiana av., **OALR**

MAINE

AUGUSTA

Turner, Oliver W......37 Stone, **OALR**

BANGOR

McCollom, Robert Lucien.....Maj. (M.C.), Dow Field, **ALR**
McQuoid, Robert Moffat.....39 Columbia, **ALR**
Whitworth, John Ewing.....49 Hammond, **ALR**

BATH

Kershner, W. E......117 Front, **OALR**

CAPE ELIZABETH

Irgens, Edwin R......
.....Maj. (M.C.), Station Hospital, Fort Williams, **ALR**

LEWISTON

Chalfers, William H......190 Bates, **ALR**

PORTLAND

Beach, S. Judd.....704 Congress, **OP**
Cummings, George O......47 Deering, **ALR**
Gordon, Charles H......46 Deering, **ALR**
Haskell, Alfred W......142 High, **OP**
Holt, E. E., Jr......723 Congress, **OP**
Johnson, Henry P......32 Deering, **ALR**
McAdams, William R......704 Congress, **OP**

WATERVILLE

Hill, Frederick T......Professional bldg., **ALR**
Hill, Howard F......179 Main, **OP**

MARYLAND

ANNAPOLIS

Chew, Wm. M., Lt. Comdr. (M.C.),
U. S. Naval Academy, Bancroft Hall, **ALR**
Macnie, John P......
.....Lt. Comdr. (M.C.), U. S. Naval Hospital, **OP**
Francis, Norton L......
.....Lt. (j. g.) (M.C.), U. S. Naval Hospital, **ALR**
Tiedke, G. E...Lt. Comdr. (M.C.), 602 Dreams Landing, **OP**
Welch, Robert S. G......86 State Circle, **OALR**
Williams, Mortimer H......
.....Lt. Comdr. (M.C.), U. S. Naval Academy, **ALR**
Wiser, Henry J., Lt Comdr. (M.C.), Sick Quarters,
Bancroft Hall, U. S. Naval Academy, **ALR**

BALTIMORE

Bagley, Cecil Hopkins.....2 E. Read, **OP**
Bordley, James Jr......4 Charlcote pl., **OALR**
Breitstein, M. Lewis.....1213 Eutaw pl., **ALR**
Dix, Harold C......405 N. Charles st., **OP**
Friedenwald, Harry.....1212 Eutaw pl., **OP A**
Friedenwald, J. S......1212 Eutaw pl., **OP**
Goldback, L. J......6 East Eager, **OP**
Goldberg, Herman Krieger.....8 E. Eager st., **OP**
Guyton, Jack Smallwood.....Johns Hopkins Hospital, **OP**
Kemler, Joseph I......1908 Eutaw pl., **OALR**
Kremen, Abraham.....2355 Eutaw pl., **OP**
Looper, Edward Anderson.....104 W. Madison, **OALR**
MacLean, Angus L......1201 N. Calvert, **OP**
McKenzie, William Raymond.....101 W. Read, **ALR**
Randolph M. Elliott.....Maj. (M.C.), 4302 Rugby rd., **OP**
Raskin, Moses.....2038 Eutaw pl., **OALR**
Small, Mary L......18 W. Read, **OP**
Snyder, Jerome.....11 E. Chase, **ALR**
Tarun, William.....104 W. Madison, **OP**
Woods, Alan C......Johns Hopkins Hospital, **OP**
Zinn, Waltman Farnsworth.....Medical Arts bldg., **ALR**

BETHESDA

Bacon, Lewis H......
.....Lt. Comdr. (M.C.), Naval Medical Center, **OP**
McGuire, Wm. P......Lt. (M.C.), U. S. Naval Hospital, **OP**
Town, A. E...Lt. Comdr. (M.C.), U. S. Naval Hospital, **OP**

BAINBRIDGE

Wible, LeRoy E......Lt. Comdr. (M.C.), Port Deposit, **ALR**

CHEVY CHASE

Rice, Carl E......6500 Maple av., **OP**

FOREST GLEN

Ridout, Geo. B......Lt. Comdr. (M.C.), U.S.N., **ALR**

MARYLAND — Continued

FORT GEORGE MEADE
Philpott I. W....Maj. (M.C.), General Hospital No. 29, ALR

FRUITLAND
Daisey, Willard Orville..... ALR

HAGERSTOWN
Smith, W. Hamilton.....314 Wareham bldg., ALR
Smoot, Merrill C.....130 Washington, ALR

HYATTSVILLE
Goad, R. R.....Lt. Comdr. (M.C.), 4211 Farragut av., OP

TACOMA PARK
Runk, R. L.....Washington Sanitarium, ALR

MASSACHUSETTS

ARLINGTON
Carroll, Walter J. E.....5 Chestnut, ALR

BEVERLY
Odeneal, Thomas Helm.....21 Broadway, OALR

BOSTON
Carter, Albert A.....419 Boylston, OP
Casten, Virgil G.....
.....Lt. Comdr. (M.C.), 520 Commonwealth av., OP
Chandler, Paul A.....5 Bay State road, OP
Chenery, W. E.....377 Commonwealth av., ALR
Chisholm, Julian F., Jr.....20 Phillips st., OP
Clarke, Samuel Tracy.....403 Commonwealth av., OP
Cline, Samuel.....416 Marlboro, ALR
Cogan, David G..Massachusetts Eye and Ear Infirmary, OP
Cowan, Clyde R.....69 Bay State road, Back Bay, ALR
DeBlois, Elizabeth.....395 Commonwealth av., ALR
Donahue, Hugh C.....520 Commonwealth av., OP
Dunphy, Edwin B...Lt. Comdr. (M.C.), 243 Charles st., OP
Easton, Mahlon T.....209 Newbury, OP
Evans, Maurice George.....416 Marlboro, ALR
Fein, Harry.....491 Commonwealth av., ALR
Fineberg, Nathan L.....475 Commonwealth av., ALR
Fred, Gustave B.....520 Beacon, ALR
Freedman, L. M.....475 Commonwealth av., ALR
Garland, Frederick E.....483 Beacon, LR
Goodale, Robert L.....330 Dartmouth, ALR
Goodall, Edwin Baker.....71 Bay State road, OP
Gorney, Arthur J.....475 Commonwealth av., ALR
Gunderson, Trygve.....Maj. (M.C.), 5 Bay State road, OP
Haley, William Thomas.....531 Beacon, ALR
Happ, L. C.....280 Beacon st., ALR
Holmes, Edgar M.....520 Commonwealth av., ALR
Hoover, Walter Boyd.....605 Commonwealth av., ALR
Inglis, Harry J.....43 Bay State road, ALR
Johns, Juanita Pearl.....313 Commonwealth av., OP
Johnson, Leighton F.....29 Bay State road, ALR
Kazanjian, V. H.....475 Commonwealth av., LR
Kelley, Vincent J.....520 Commonwealth av., ALR
Kleinert, Margaret Noyes.....82 Commonwealth av., ALR
Lancaster, W. B.....520 Commonwealth av., OP
Lewis, J. P.....479 Commonwealth av., ALR
Lovesey, Burton E.....76 Bay State road, ALR
Lurie, Moses H.....483 Beacon, ALR
Meltzer, Philip E.....20 Charlesgate West, ALR
Messenger, Harry K.....313 Commonwealth av., OP
Mills, Chester R.....520 Commonwealth av., ALR
Mueller, Werner.....412 Beacon, ALR
Poirier, George H.....60 Bay State road, ALR
Porter, Charles T.....520 Commonwealth av., ALR
Quincy, J. E.....270 Commonwealth av., ALR
Regan, James J.....520 Commonwealth av., OP
Regan, William F.....19 Bay State road, ALR
Richards, L. G.....319 Longwood av., ALR
Richardson, John R.....395 Commonwealth av., ALR
Rowland, W. D.....84 Commonwealth av., OP
Sachs, Benjamin.....520 Beacon, OP
Schall, LeRoy A.....243 Charles st., ALR
Skirball, J. J.....353 Commonwealth av., OP
Sloane, Albert E.....416 Marlborough, OP
Terry, Theodore L.....140 Marlborough, OP
Thompson, Peter H.....398 Commonwealth av., OP
Thorne, Fred S.....270 Commonwealth av., OP
Tingley, Louisa Paine.....416 Marlborough, OP
Tobey, George L., Jr.....403 Commonwealth av., ALR
Verhoeff, Frederick.....395 Commonwealth av., OP
Wells, David W.....31 Bay State road, OP
Wilkins, Samuel Henry.....270 Commonwealth av., OP

BROCKTON
Jones, Edward Earl.....141 West Elm, ALR
Norbonne, Joseph J.....231 Main, OP

BROOKLINE
Gillespie, Elmer H.....1101 Beacon, ALR

CAMBRIDGE
Butler, Edmund John.....25 Garden st., ALR

CHICOPEE FALLS
Dearmin, R. M.....Station Hospital, Westover Field, ALR

CLINTON
Monahan, J. A.....181 Chestnut, OALR
O'Toole, Francis Austin.....181 Chestnut, ALR

FALL RIVER
Best, Lynwood K.....151 Rock, OP
Ryder, D. R.....151 Rock, ALR

FORT DEVENS
Joyce, W. M.....Maj. (M.C.), New Station Hospital, ALR
Riseman, Benjamin.....
.....Capt. (M.C.), Lowell General Hospital, ALR
Schultz, Abraham.....
.....Capt. (M.C.), Lowell General Hospital, OP

FRAMINGHAM
Dinan, Thomas Edward.....132 Union av., ALR
Sails, Frederick H.....132 Union av., OP
Tomb, Everett H.....Maj. (M.C.), 46 Lexington, ALR

GARDNER
Bone, Herman D.....19 Pleasant, OALR

GLOUCESTER
Odeneal, Thomas H.....No. 9 Middle, OALR

GREENFIELD
Powers, John T. H.....57 Federal st., ALR

HAVERHILL
Birdsall, Clarence H.....30 Summer, ALR
Capeles, T. F.....191 Merrimack, ALR
Kapp, Henry.....50 Merrimack, ALR
Shlosberg, Frank Ronald.....116 Emerson, ALR
Trull, Alfred C.....3 Washington square, OP

HOLYOKE
Burke, John R.....207 Elm, OALR
Halton, Edward P.....245 Maple, ALR

LAWRENCE
Byrne, Harry V.....301 Essex, OP

LOWELL
Berman, Philip G.....174 Central, OP
Leahey, Brendan D.....9 Central, OP
Leahey, George A.....45 Merrimack, OP
Wiggin, William Irving.....202 Central, ALR

MASSACHUSETTS — Continued

LYNN
- Jones, E. W. — 44 Atlantic, OP A
- McIntire, Frederic J. — 63 N. Common, ALR
- O'Reilly, W. F. — 44 S. Common, OALR
- Rafferty, George E. — 173 N. Common, ALR

MALDEN
- French, Robert Weller — 181 Pleasant, OP
- Haire, Paul G. — 6 Pleasant, OP

MARBLEHEAD
- Mosher, Harris P. — 127 Front, ALR

MEDFORD
- Haley, Paul J. D. — 333 Winthrop, ALR

MELROSE
- DeWolf, Charles W. — 8 Porter, ALR

NORTHAMPTON
- Collins, J. D. — 187 Main, ALR
- Dow, Frank E. — 4 West, OP
- Seelig, Charles A. — Lt. (M.C.), U.S.N.R., 10 Dryads Green, ALR

PALMER
- Moore, G. A. — Box 471, OALR

PITTSFIELD
- Porter, Byron H. — 18 Bank Row, ALR
- Thompson, G. H. — 74 North, OALR

QUINCY
- Gilmore, Robert O. — 1159 Hancock, ALR

SALEM
- Adams, John G. — 24 Winter, ALR
- Hennessey, W. W. — 333 Essex, OALR
- Tolman, Henry — 136 Federal, ALR

SOMERVILLE
- Solo, Adrian — 174 Highland av., ALR

SPRINGFIELD
- Boss, Eugene G. — 1537 Main, OALR
- Byrnes, H. F. — 67 Chestnut, OALR
- Gaudreau, Honore E. — 293 Bridge, OP
- Goodell, William — 121 Chestnut, ALR
- Irwin, Vincent J., Jr. — 1634 Main, OALR

TAUNTON
- Cowan, Thomas H. — Capt. (M.C.), 220 High st., OP

WALTHAM
- Lewis, J. P. — 681 Main, ALR
- Mackenzie, Roland C. — 30 Grant, OP

WORCESTER
- Berry, Gordon — 36 Pleasant, ALR
- Caron, Armand L. — 36 Pleasant, ALR
- Croissant, Charles A. — 390 Main, ALR
- Cross, A. E. — 390 Main, OP
- Estabrook, Charles T. — 10 Algonquin road, OALR
- Gibby, Harold James — 27 Elm, ALR
- Goodspeed, Harry B. — 36 Pleasant, ALR
- Holzer, William F. — 36 Pleasant, OP
- Messier, A. Eugene — 390 Main, OALR
- Murphy, William E. — 390 Main, ALR
- Rice, John E. — 390 Main, OALR
- Tegelberg, Julius J. — Lt. Comdr. (M.C.), 12 Monterey rd., ALR
- Whitney, Percy T. — 390 Main, OP

MICHIGAN

ALPENA
- Shilling, R. L. — Capt. (M.C.), Base Hospital, Army Air Base, ALR

ANN ARBOR
- Barr, Albert S. — First Nat'l Bank bldg., OP
- Fralick, F. Bruce — First Nat'l Bank bldg., OP
- Furstenberg, A. C. — First Nat'l Bank bldg., ALR
- Maxwell, James H. — First Nat'l Bank bldg., ALR
- Teed, R. Wallace, Lt. Comdr. (M.C.), 410 Highland rd., ALR

BATTLE CREEK
- Barkhorn, Charles W. — Maj. (M.C.), Percy Jones General Hospital, ALR
- Dodge, Warren M., Jr. — Maj. (M.C.), 186 N. Wabash, OP
- Fraser, R. H. — 25 W. Michigan av., ALR
- Haughey, Wilfrid H. — Post bldg., ALR
- Sleight, R. D. — Security Nat'l Tower, OP
- Wencke, Carl G. — 1015 Security Tower, ALR

BAY CITY
- Baker, Charles H. — Bay City Bank bldg., OALR
- Criswell, Robert Harold — Phoenix bldg., OP
- Kessler, Mana — 311 Center av., ALR
- Sherman, R. Newton — Phoenix bldg., OALR

BENTON HARBOR
- Cawthorne, Harold Jesse — 239 Pipestone, ALR
- Westervelt, H. O. — 239 Pipestone, ALR

BIRMINGHAM
- Ferris, Ralph G. — 55 W. Maple av., ALR

COLDWATER
- Thomas, James Alanson, Southern Michigan Bank bldg., ALR

DETROIT
- Amberg, Emil — 1244 W. Boston blvd., ALR
- Barnett, Saul E. — 25 Parsons, ALR
- Beattie, Robert — David Whitney bldg., OALR
- Begle, Howell L. — 2730 East Jefferson av., OP
- Bentley, Neil I. — David Whitney bldg., OALR
- Birch, John R. — Maccabees bldg., ALR
- Brown, Audrey O. — 5057 Woodward av., OALR
- Brunk, Clifford F. — 7815 E. Jefferson av., ALR
- Campbell, Duncan A. — David Whitney bldg., OALR
- Carter, John M. — David Whitney bldg., ALR
- Carter, Leland Fields — David Whitney bldg., OP
- Clarke, George L. — 910 Chalmers, ALR
- Clifford, T. P. — In Service, David Whitney bldg., OALR
- Clifford, T. P. — In Service (address unknown), OALR
- Cohoe, D. A. — 13535 Woodward av., ALR
- Cooper, Edmond L. — 414 David Whitney bldg., OP
- Crossen, Henry F. — David Whitney bldg., ALR
- Croushore, James E. — Maj. (M.C.), 2045 Edison, ALR
- Defnet, William A. — 28 Adams av., West, ALR
- Dill, J. Lewis — Henry Ford Hospital, ALR
- Dowling, Harvey E. — Eaton Tower, OP
- Dunlap, Henry Arthur — 7815 E. Jefferson av., OP
- Gemeroy, Joseph C. — Henry Ford Hospital, OALR
- Gleason, John E. — David Whitney bldg., ALR
- Glowacki, Ben Francis — Maccabee bldg., OALR
- Gonne, William S. — David Whitney bldg., ALR
- Goux, Raymond Salot — David Whitney bldg., OALR
- Hale, Arthur Stanley — Eaton Tower, OALR
- Hammond, Arthur — David Whitney bldg., ALR
- Harrell, Vuss — Hofman bldg., ALR
- Hartgraves, Hallie — 28 West Adams av., OP
- Heath, Parker — David Whitney bldg., OP

MICHIGAN — Continued

Hughes, Ray W. — David Whitney bldg., OALR
Joinville, Euclide V. — Stroh bldg., ALR
Keating, T. F. — Maccabee bldg., OALR
Lamberson, Frank Abrom — 19600 Grand River, ALR
Lepard, Cecil W. — David Whitney bldg., OP
McCullough, Lester E. — 1553 Woodward av., OP
MacFarlane, Howard W. — David Whitney bldg., ALR
May, Frederick Tracy — Maj. (M.C.), 16637 Parkside, OP
Mercer, R. E. — 126 Monterey, LR
Mond, Edward — Eaton Tower, ALR
Morton, J. B. — 90 E. Warren av., OALR
Munson, F. T. — 2201 E. Jefferson av., ALR
O'Hora, James Timothy — General Motors bldg., ALR
Parker, Walter R. — David Whitney bldg., OP
Peirce, Howard W. — David Whitney bldg., ALR
Pino, Ralph H. — David Whitney bldg., OP
Pittman, John E. — David Whitney bldg., OP
Poos, E. E. — Fisher bldg., OALR
Potter, Willis A. — 7450 Grand River av., ALR
Proctor, Bruce — Eaton Tower, ALR
Robb, James Milton — David Whitney bldg., ALR
Ryerson, F. L. — Fine Arts bldg., OALR
Scarney, Herman D. — Fisher bldg., OP
Shurly, Burt R. — 62 Adams av. West, ALR
Simpson, H. Lee — David Whitney bldg., ALR
Summers, William S. — David Whitney bldg., OALR
Sutherland, Jacob Marion — Fisher bldg., ALR
Vossler, Albert E. — David Whitney bldg., OALR
Weaver, Delmar Franklin — Henry Ford Hospital, ALR
Wendel, J. S. — David Whitney bldg., ALR
Whitney, E. L. — Henry Ford Hospital, OP
Wilkinson, A. P. — 3001 W. Grand blvd., ALR
Willson, W. W. — 1553 Woodward av., OALR
Woodry, Norman Lee — 10 Witherell, ALR
Woodworth, William P. — 2501 W. Grand blvd., OALR

FLINT

Hubbard, William B. — Maj. (M.C.), 1205 Maxine st., OALR
Smith, Eugene C. — Union Industrial bldg., OALR
Stevenson, William W. — Kresge bldg., OALR
White, Herbert T. — National bldg., OP

FORT BRADY

Good, Richard P. — Maj. (M.C.), Station Hospital, ALR

GRAND RAPIDS

Dean, Alfred — Medical Arts bldg., OP
Dewar, Murray M. — Medical Arts bldg., OP
Doran, Frank — Metz bldg., ALR
Gilbert, Ralph H. — Metz bldg., OP
Heetderks, D. R. — Medical Arts bldg., ALR
Laird, Robert G. — Metz bldg., OP
McRae, J. H. — Medical Arts bldg., OP
Moll, Arthur M. — Powers Theatre bldg., ALR
Phillips, James William — Medical Arts bldg., OP
Smith, Ferris — Blodgett Medical bldg., ALR
Snapp, Carl F. — Medical Arts bldg., ALR

GROSSE POINT

Lathrop, Frank D. — Maj. (M.C.), 541 Lincoln road, ALR
Merkel, Charles C. — 85 Kercheval av., ALR

HOLLAND

DeVries, Harold G. — 36 East 8th, ALR

IONIA

Sedgwich, O. W. — OALR

IRON MOUNTAIN

Boyce, G. H. — First Nat'l Bank bldg., OALR
Menzies, Clifford G. — 733 Woodward av., ALR

JACKSON

Hardie, George C. — 290 W. Michigan av., OALR
McGarvey, William Edward — Jackson City Bank bldg., OALR
Winter, George E. — 117 W. Michigan av., OALR

KALAMAZOO

Fast, Ralph B. — 136 E. Michigan av., ALR
Fulkerson, C. B. — 107 W. Michigan av., OALR
Grant, Fred E. — Kalamazoo Nat'l Bank bldg., OALR
Marshall, Don — American Nat'l Bank bldg., OP
Wilbur, Edward P. — American Nat'l Bank bldg., OALR

LANSING

Behen, William C. — 535 S. Capitol av., ALR
Burdick, Austin F. — P. O. Drawer 1378, OALR
Ellis, C. Ward — 215 N. Walnut, ALR
McGillicuddy, Oliver B. — 124 W. Allegan, ALR
Wetzel, John O. — Olds Tower bldg., OP

MENOMINEE

Jones, William S. — 521 Sheridan road, OALR

MIDLAND

Pike, Melvin H. — Reinhart bldg., OALR

MT. CLEMENS

Croman, Joseph M., Jr. — 115 Cass av., OALR

MUSKEGON

Mandeville, Clifford Bagley — Hackley Union Bank, ALR

NILES

Moore, Thomas Scott — 107 N. 2nd, OP

OWOSSO

Pochert, Rolland C. — Matthews bldg., OALR

PONTIAC

Larson, Bertil T. — Riker bldg., ALR
Riker, Aaron D. — Riker bldg., OP

PORT HURON

Fraser, Robert — 940 Military, ALR

RIVER ROUGE

Flaherty, Norman W. — 10593 W. Jefferson av., OP

ST. JOSEPH

McDermott, John J. — 505 Pleasant st., OALR

SAGINAW

Cady, F. J. — 402 S. Jefferson av., OALR
Cortopassi, Andre J. — 324 S. Washington av., ALR
Gomon, Louis D. — Eddy bldg., OP
Howell, Donald M. — 2112 Gratiot av., ALR
McKinney, A. R. — 330 S. Washington av., OP

SELFRIDGE FIELD

Calvert, Raymond R. — Maj. (M.C.), OP
Sims, J. Lawrence — Station Hospital, ALR

MINNESOTA

ALBERT LEA

Folken, Frank Garrett — 210 S. Broadway, OALR

BEMIDJI

Garlock, Arthur Varney — 217½ 3rd, OP

CROOKSTON

Morley, George A. — Crookston State Bank bldg., OP
Oppegaard, C. L. — 220 S. Broadway, ALR
Parsons, J. G. — Crookston Clinic, OALR

MINNESOTA — Continued

DULUTH

Armstrong, E. Leslie.....Medical Arts bldg., **ALR**
Athens, Alvin G......Medical Arts bldg., **OP**
Hilding, Anderson C......Medical Arts bldg., **OALR**
Knapp, F. N......Medical Arts bldg., **OALR**
Olson, Archie.....Medical Arts bldg., **OP**
Shastid, T. H......202 W. Superior, **OP**
Tilderquist, D. L......Medical Arts bldg., **OALR**
Winter, John A......205 W. 2nd, **OALR**

FERGUS FALLS

Combacker, Leon C......101½ Lincoln av. West, **OP**

HIBBING

Morsman, L. W......Morsman Clinic, **OP**
Sinamark, Andrew.....Power bldg., **OALR**

MINNEAPOLIS

Boies, Lawrence R......Medical Arts bldg., **ALR**
Borgeson, E. J......Medical Arts bldg., **OP**
Bryant, Frank L......1600 W. Lake, **ALR**
Cabot, C. M......Medical Arts bldg., **ALR**
Camp, Walter E......Medical Arts bldg., **OALR**
Clark, H. S......2615 Park av., **OALR**
Cooperman, Harold O......Medical Arts bldg., **OP**
Curtin, John F......Medical Arts bldg., **ALR**
Dahl, Elmer O......Medical Arts bldg., **ALR**
Dorge, Richard Irving.....1111 Nicollet av., **OALR**
Fink, Leo W......Medical Arts bldg., **ALR**
Fink, Walter H......Medical Arts bldg., **OP**
Garten, Joseph Lambert.....Medical Arts bldg., **ALR**
Hansen, Erling W......Medical Arts bldg., **OALR**
Hoffman, Walter Lees.....Medical Arts bldg., **OP**
Howard, Wm. H......310 Physicians and Surgeons bldg., **OALR**
Hymes, Charles.....Medical Arts bldg., **OP**
Koller, Hermann M......Medical Arts bldg., **OP**
Koller, Louis R......Medical Arts bldg., **ALR**
Lindberg, Vernon Leslie.....
.....Capt. (M.C.), Physicians and Surgeons bldg., **OP**
Litchfield, J. T......Medical Arts bldg., **OALR**
Loomis, E. A......Medical Arts bldg., **OALR**
McGeary, George E......Medical Arts bldg., **ALR**
Macnie, J. S......Medical Arts bldg., **OALR**
Matthews, Justus.....Medical Arts bldg., **LR**
Newhart, Horace.....Medical Arts bldg., **ALR**
Oberg, C. M......Syndicate bldg., **OALR**
Patterson, W. E......Medical Arts bldg., **OALR**
Pfunder, Malcom C......Medical Arts bldg., **ALR**
Phelps, Kenneth A......Medical Arts bldg., **OALR**
Pollock, F. J......Lt. (M.C.), U.S.N.R., Roanoke bldg., **ALR**
Pratt, Fred J......Physicians and Surgeons bldg., **OALR**
Ricketts, Floyd B......Veterans Hospital, **ALR**
Ricketts, Floyd B......Veterans Hospital, **ALR**
Sandt, Karl E......Medical Arts bldg., **OP**
Schwartz, Virgil Joseph.....Medical Arts bldg., **ALR**
Smith, Arthur E......Medical Arts bldg., **OALR**
Stanford, Charles E......Medical Arts bldg., **OP**
Strout, G. Elmer.....1009 Nicollet av., **OALR**
White, C. H......Lt. Comdr. (M.C.), Naval Aviation Base, **ALR**

NEW ULM

Fritsche, Theodore R......Fritsche bldg., **OALR**
Reineke, George F......Fritsche bldg., **OALR**

RED WING

Brusegard, James F......414 Main, **OP**

ROCHESTER

Benedict, William L......Mayo Clinic, **OP**
Figi, Frederick A......Mayo Clinic, **LR**
Hallberg, Olav Erik.....Mayo Clinic, **ALR**
Hempstead, Bert E......Mayo Clinic, **ALR**
Lillie, Harold I......Mayo Clinic, **ALR**
New, Gordon B......Mayo Clinic, **L**
Prangen, Avery de H......Mayo Clinic, **OP**
Rucker, C. W......Mayo Clinic, **OP**
Simonton, Kinsey Macleod.....Mayo Clinic, **ALR**
Wagener, Henry P......Mayo Clinic, **OP**
Williams, Henry Lane.....Mayo Clinic, **ALR**

ST. CLOUD

Gaida, Joseph Benedict.....St. Mary's bldg., **OP**
Wenner, W. T......St. Mary's bldg., **OP**

ST. PAUL

Binger, H. E......Lowry Medical bldg., **OALR**
Boeckmann, Egil.....Lowry Medical bldg., **OALR**
Brown, John C......Medical Arts bldg., **OALR**
Burch, Frank E......754 Linwood place, **OP**
Connor, Charles E......Lowry Medical bldg., **ALR**
Edwards, Thomas J......330 Lowry Medical bldg., **OP**
Fogarty, Charles W......372 St. Peter, **OALR**
Grant, H. W......Lowry Medical bldg., **OP**
Hochfilzer, John J......Lowry Medical bldg., **ALR**
Kennedy, William A......372 St. Peter, **OP**
Koch, Ferdinand L. P......408 St. Peter st., **OP**
Larsen, Carl L......Lowry Medical bldg., **OALR**
Leavenworth, R. O......Lowry Medical bldg., **OP**
Nelson, Louis A......Lowry Medical bldg., **OP**
O'Reilley, Bernard E......Lowry Medical bldg., **OP**
Rothschild, Harold J......Lowry Medical bldg., **OALR**
Shellman, J. L......Lowry Medical bldg., **OP**
Tracht, Robert R......Lowry bldg., **OP**
Wheeler, Merritt W......Lowry Medical bldg., **OALR**
Wold, K. C......Lowry Medical bldg., **OALR**

WINONA

Risser, Edward D......Choate bldg., **OALR**
Walker, George H......172 Main, **OALR**

WORTHINGTON

Stanley, C. R......508 Tenth, **ALR**

MISSISSIPPI

BILOXI

Boden, Worthey C......Capt. (M.C.), Keesler Field, **ALR**

CAMP SHELBY

Jenkins, Albert H......
.....Capt. (M.C.), 98th Evacuation Hospital, **ALR**

CAMP VAN DORN

Irwin, Willard J......Capt. (M.C.), Station Hospital, **ALR**

CLARKSDALE

Wilkins, Ellis LeRoy.....McWilliams bldg., **ALR**

CORINTH

Hamrick, DeWitt.....601 Jackson, **ALR**

GREENVILLE

Montgomary, D. Cameron.....Weinberg bldg., **ALR**

GREENWOOD

Pegues, James Cary.....301 Howard st., **ALR**

GRENADA

Caruthers, Samuel Brooks.....Grenada Clinic, **OALR**

MISSISSIPPI — Continued

GULFPORT

McWilliams, Charles A.....1300 27th av., **ALR**
Sharp, Benjamin S.....Maj. (M.C.), Station Hospital, **OALR**
Wilson, Alfred....Maj. (M.C.), 1518 20th av., **OP**

JACKSON

Adkins, George E.....Jackson Infirmary, **ALR**
Anderson, Ross Epting....Standard Life bldg., **ALR**
Hagaman, Van Dyke....Lamar Life bldg., **ALR**
Harris, Robin....964 N. State, **ALR**
Hollowell, Frederick Dixon....Lamar Life bldg., **ALR**
Hughes, William Lauch....Lamar Life bldg., **ALR**
McNair, Stirling Sharp....
....Lt. Comdr. (M.C.), 1228 Monroe st., **OP**

KEESLER FIELD

Hall, Sobisca S.....Maj. (M.C.), Station Hospital, **ALR**
McAlpine, P. T.....Capt. (M.C.), Station Hospital, **OP**

NATCHEZ

Gaudet, Lucien S.....405 Franklin, **OALR**

OXFORD

Guyton, B. S.....512 Van Buren av., **OALR**

VICKSBURG

Edwards, C. J.....First Nat'l Bank bldg., **OALR**
Jones, Edley H.....1301 Washington, **OALR**

MISSOURI

CAMP CROWDER

Greenberg, Maynard M.....
....Capt. (M.C.), Station Hospital, **OP**
Johnson, Malcolm R.....
....Maj. (M.C.), 140th Station Hospital, **ALR**

CAPE GIRARDEAU

Tygett, Glenn J.....215 N. Sprigg, **ALR**

CARTHAGE

Powers, Everett....305 S. Main, **OALR**

COLUMBIA

Sneed, Carl M.....Exchange bldg., **OALR**

FORT LEONARD WOOD

Arbuckle, Millard F.....Col. (M.C.), Station Hospital, **ALR**
Simons, Leander T.....Maj. (M.C.), Station Hospital, **OP**

HANNIBAL

Hornback, George A.....500 Broadway, **ALR**

KANSAS CITY

Allen, Charles E.....Waldheim bldg., **OALR**
Altringer, A. N.....Professional bldg., **ALR**
Beal, Homer A.....In Service, Professional bldg., **ALR**
Beil, J. W.....Bryant bldg., **OALR**
Black, W. Byron....Professional bldg., **ALR**
Blakesley, T. S.....Professional bldg., **OALR**
Connell, Evan Shelby....Commerce bldg., **OP**
Curdy, R. J.....1102 Grand av., **OP**
Curran, Desmond....Federal Reserve Bank bldg., **OP**
Curran, E. J.....Federal Reserve Bank bldg., **OP**
Curran, Kevin....925 Grand av., **OP**
Dixon, O. Jason....Professional bldg., **ALR**
Dyar, Edwin William....
....Lt. (M.C.), U.S.N.R., 649 W. 70th st., **OP**
Eubank, Ambrose E.....Bryant bldg., **OALR**
Foster, Hal....(Retired), Hyde Park Hotel, **ALR**
Gilliland, O. S.....Professional bldg., **ALR**
Howard, J. W.....(Retired), Residence 6026 McGee, **OALR**
Howard, John Clair....
....Lt. (j. g.) (M.C.), Professional bldg., **ALR**
Keith, W. E.....Professional bldg., **OALR**
Knight, John S.....Professional bldg., **ALR**
Lemoine, A. N.....Rialto bldg., **OP**
Lux, Paul....Bryant bldg., **ALR**
McAlester, Andrew W., Jr.....Bryant bldg., **OP**
McKee, Joseph W.....Professional bldg., **OP**
Miller, Hugh....Argyle bldg., **OALR**
Myers, John L.....Shukert bldg., **OALR**
Roberts, S. E.....Professional bldg., **ALR**
Shoemaker, Ferdinand....3232 Harrison, **OALR**
Simpson, M. B.....Professional bldg., **ALR**
Teall, Raymond E.....Professional bldg., **ALR**
Thomason, H. E.....Professional bldg., **ALR**
Trowbridge, Barnard Cole....Commerce bldg., **ALR**
Yazel, Herman E.....Rialto bldg., **OALR**

KIRKWOOD

McQuiston, Ralph J.....Maj. (M.C.), 203 Barter av., **ALR**

JEFFERSON BARRACKS

Baer, Alvin Julius....Maj. (M.C.), Station Hospital, **OP**

ROLLA

Boiler, W. F.....Rolla Gardens, **OALR**

ST. JOSEPH

Minton, William H.....Corby bldg., **OALR**

ST. LOUIS

Alden, Arthur M.....Frisco Bldg., **ALR**
Alvis, Bennett Y.....7011 Washington blvd., **OP**
Alvis, Edmund B.....7011 Washington blvd., **OP**
Bardenheier, F. G. A.....Frisco bldg., **ALR**
Beisbarth, Carl....3720 Washington blvd., **OP**
Birsner, Louis J.....6045 Waterman av., **ALR**
Black, W. D.....Metropolitan bldg., **ALR**
Boemer, L. C., Maj. (M.C.), U.S.A.,
In Service (address unknown), **ALR**
Brown, Clyde O.....3720 Washington blvd., **ALR**
Bryan, William T. K.....3720 Washington blvd., **ALR**
Burns, S. S.....Missouri Theatre bldg., **ALR**
Charles, Joseph W.....Humboldt bldg., **OP**
Costen, J. B.....Beaumont Medical bldg., **ALR**
Dean, Lee W., Jr.....University Club bldg., **ALR**
Dobbs, Otto Ross....
....Lt. Comdr. (M.C.), 9927 Riverview drive, **ALR**
Dyer, C. P.....Humboldt bldg., **OP**
Eber, Carl Theodore....Carleton bldg., **OP**
Elmer, Charles Eugene....462 N. Taylor av., **ALR**
Elz, Jule Theodore....3606 Gravois blvd., **OP**
Fineberg, Maxwell....462 N. Taylor av., **ALR**
Glick, Harry N.....3115 S. Grand blvd., **ALR**
Gorrilla, Laurence Vincent....3115 S. Grand blvd., **OP**
Green, John....3720 Washington blvd., **OP**
Green, M. L.....Missouri Theatre bldg., **OP**
Gundelach, C. A.....University Club bldg., **ALR**
Hansel, French Keller....634 N. Grand av., **ALR**
Hardesty, J. F.....Missouri Theatre bldg., **OP**
Hardy, W. F.....Humboldt bldg., **OP**
Hildreth, H. Rommel....Metropolitan bldg., **OP**
Hobart, Carl....Metropolitan bldg., **OP**
Hoose, C. H. A.....3115 S. Grand blvd., **OP**
Hourn, George E.....University Club bldg., **ALR**
Howard, Harvey J.....Park Plaza Hotel, **OP**
James, William M.....508 N. Grand av., **OP**
Jones, Vincent L.....Missouri Theatre bldg., **OALR**
Kelley, Isaac D., Jr.....3720 Washington blvd., **ALR**
Lange, Adolph Charles....3903 Olive, **OP**
Leavy, C. A.....Metropolitan bldg., **ALR**

MISSOURI — Continued

Luedde, W. H.....Metropolitan bldg., **OP**
Lyman, Harry W.....308 N. 6th, **ALR**
McMahon, Bernard John....Missouri Theatre bldg., **ALR**
Mason, Roy Earl....Frisco bldg., **OP**
Mattis, Robert Dean...Lt. (M.C.), 6223 Southwood av., **OP**
Meinberg, William H.....906 Olive, **OP**
Miller, H. Edward....634 N. Grand blvd., **ALR**
Milligan, Roy H.....3720 Washington blvd., **ALR**
Morman, William Daniel....3507 Franklin av., **ALR**
Myers, E. Lee....Wall bldg., **ALR**
Nemours, Paul Roland....1506 Hodiamont av., **ALR**
Payne, R. J.....University Club bldg., **ALR**
Post, Lawrence T.....Metropolitan bldg., **OP**
Post, M. Hayward....Metropolitan bldg., **OP**
Potter, Allen B.....Pasteur Medical bldg., **ALR**
Proetz, A. W.....Beaumont bldg., **ALR**
Reim, Hugo....Metropolitan bldg., **OP**
Ryan, L. M.....Wall bldg., **ALR**
St. John, Dewey....Metropolitan bldg., **ALR**
Sanders, Theodore E.....Metropolitan bldg., **OP**
Sauer, W. E.....Beaumont Medical bldg., **ALR**
Scholz, R. P.....Metropolitan bldg., **ALR**
Schwartz, Frederick O.....508 N. Grand blvd., **OP**
Senseney, Eugene T.....Lister bldg., **ALR**
Senturia, Ben H.....Capt. (M.C.), 4475 W. Pine blvd., **ALR**
Shahan, W. E.....Metropolitan bldg., **OP**
Shoemaker, W. A.....220 N. Kingshighway blvd., **OP**
Smit, William M.....5285 Washington blvd., **ALR**
Stutsman, Albert C.....539 N. Grand blvd., **ALR**
Tooker, C. W.....Carleton bldg., **OP**
Walsh, Theodore E.....640 S. Kingshighway blvd., **ALR**
Westlake, Samuel B.....Beaumont Medical bldg., **ALR**
Wood, V. Visscher....Beaumont Medical bldg., **ALR**
Woodruff, Frederick E.....Metropolitan bldg., **OP**

SPRINGFIELD

Bailey, Harold....Landers bldg., **OALR**
Burch, Edward P.
....Maj. (M.C.), O'Reilly General Hospital, **OP**
Cheek, Carey....Medical Arts bldg., **OP**
Hauser, I. Jerome
....Maj. (M.C.), O'Reilly General Hospital, **ALR**
Klingner, Thomas O.....Medical Arts bldg., **OALR**
Smith, Carlie Souter....Holland bldg., **OALR**

TRENTON

Ambrose, Elmer C.....910 Main, **OALR**

WEBSTER GROVES

Dean, L. W.....1125 N. Berry road, **ALR**
Dyer, Clyde P.....Garelock bldg., **OP**

MONTANA

ANACONDA

Dunlap, Lawrence G.....101 Main, **OALR**

BILLINGS

Morledge, Roy V.....Hart-Albin bldg., **ALR**
Morrison, Wm. R.....Hart-Albin bldg., **OALR**

GREAT FALLS

Hurd, Fritz D.....Medical Arts bldg., **ALR**

MILES CITY

Rowen, Ernest Henry....Box 570, **ALR**

MISSOULA

Marshall, William J.....Montana block, **ALR**
Murphy, Edward S.....Lt. Col. (M.C.), Dixon bldg., **OP**
Stephan, Lewis Behymer....Western Montana Clinic, **OP**

ROUNDUP

Lewis, George A.....213 Main, **OALR**

NEBRASKA

COLUMBUS

Campbell, C. H.....2514 Thirteenth, **OALR**

FREMONT

Heine, Lyman H.....635 N. Main, **ALR**
Heine, William Howard....Odd Fellow bldg., **ALR**

GRAND ISLAND

Martin, Roy David....Masonic bldg., **ALR**

LINCOLN

Black, Paul....Stuart bldg., **OALR**
Briggs, Henry H., Jr.....Maj. (M.C.), 3064 Puritan st., **OP**
Brooks, Earl B.....128 N. 13th st., **OALR**
Furgason, A. P.....Richards block, **OALR**
Hompes, J. J.....Sharp bldg., **OALR**
Sandereon, David D.....Stuart bldg., **OALR**
Thomas, James W.....Sharp bldg., **OALR**
Zomer, S. G.....Stuart bldg., **OALR**

NEBRASKA CITY

Stonecypher, D. D.....107 S. 9th, **ALR**

NORFOLK

Gadbois, Arthur E.....1219 Koenigstein av., **OALR**

OMAHA

Bushman, L. B.....City Nat'l Bank bldg., **OALR**
Cassidy, W. A.....Medical Arts bldg., **ALR**
Cecha, Albin H.....4437 Woolworth av., **OP**
Coakley, Leo P.....Medical Arts bldg., **ALR**
Davis, John Calvin, Jr.....425 Aquila court, **OALR**
Gifford, Harold....Medical Arts bldg., **OP**
Hahn, William N.....City Nat'l Bank bldg., **OP**
Haney, William P.....Medical Arts bldg., **OALR**
Judd, J. H.....Medical Arts bldg., **OP**
Kully, Herman E.....Medical Arts bldg., **ALR**
Morrison, Wm Howard....Medical Arts bldg., **OP**
Potts, J. B.....3817 Dewey av., **ALR**
Romonek, Philip L.....Medical Arts bldg., **OALR**
Rubendall, C.....Medical Arts bldg., **OALR**
Stokes, William H.....Medical Arts bldg., **OP**
Swab, Charles M.....Medical Arts bldg., **OP**
Watke, Frederic Martin....Medical Arts bldg., **ALR**

NEVADA

LAS VEGAS

Richman, Frances....750 Fremont st., **OP**
Thorburn, O. L.
....Maj. (M.C.), Station Hospital, Gunnery School, **ALR**

RENO

Creveling, Earle LaMont....17 N. Virginia st., **ALR**
Fuller, John Andre....First Nat'l Bank bldg., **OALR**

NEW HAMPSHIRE

BERLIN

Ingalls, Raymond George....St. Louis Hospital, **OP**

NEW HAMPSHIRE — Continued

CONCORD
MacMillan, Andrew L., Jr. — 46 Pleasant, ALR

DERRY
Pearson, Ovid — 18 Pleasant st., OALR

DOVER
Reid, Thomas F. — Eight 3rd, ALR

HANOVER
Burlan, Hermann M. — The Eye Clinic, OP
Murtagh, John Anthony — Hitchcock Clinic, ALR
Roper, Kenneth Lawrence — Clinical Division, Dartmouth Eye Institute, OP

KEENE
Ford, LeRoy Sargent — 30 School st., ALR

MANCHESTER
Monette, C. J. — 814 Elm, ALR
Rahmanop, Walter B. — 1426 Elm, ALR
Togus, Leopold T. — 967 Elm, ALR

NASHUA
Marcotte, Raymond H. — 196 Main, ALR
Nutter, C. F. — 16 Amherst, OP

PORTSMOUTH
Allen, Cary D. — Comdr. (M.C.), U. S. Naval Prison, Navy Yard, ALR
Cordray, David P. — Lt. Comdr. (M.C.), U. S. Naval Hospital, ALR

NEW JERSEY

ASBURY PARK
Baeseman, R. Winfield — 501 Grand av., OALR
Fisher, James A. — Jersey Central bldg., OALR
Upham, Helen F. — 305 Third av., OALR

ATLANTIC CITY
Alfaro, V. R. — Capt. (M.C.), Station Hospiral, ALR
Charlton, C. Coulter — 124 S. Illinois av., ALR
Dalton, Simon E. — 117 S. Illinois av., ALR
Gordon, Benjamin Lee — 1616 Pacific av., OP
Steinberg, M. R. — Maj. (M.C.), A.A.F.T.T.C., B.T.C. No. 7, ALR

BOUND BROOK
Levy, Abram — 507 Church, OP

CAMDEN
Hirst, Ernest Reed — 634 Federal, ALR
Kline, Oram R. — 514 Cooper, ALR
Meyer, George P. — 410 Haddon av., OP
Ruttenberg, Max — 303 Cooper, ALR
Shemeley, William G., Jr. — 7 Haddon av., OALR
Shipman, James S. — 514 Cooper, OP
Shope, Pierce — 511 Cooper, OP

CAMP KILMER
Appelbaum, Alfred — Maj. (M.C.), Station Hospital, OP

CAPE MAY
Segal, Samuel, Jr. — Lt. Comdr. (M.C.), U. S. Naval Base, ALR

EAST ORANGE
Buvinger, Charles W. — 50 Washington, OALR
Campbell, William — 144 S. Harrison, ALR
Cregar, John S. — 150 Harrison, OP
Sherman, Arthur E. — 144 S. Harrison, OP

ELIZABETH
Ehrlich, Max — Capt. (M.C.), 721 W. Broad, ALR
Gadomski, Casimir Francis — Lt. (M.C.), 331 S. Broad st., OP
Kapp, Carl G. — 440 Westminster av., ALR

GLEENWOOD
Worcester, George F. — 220 Engle, ALR

FORT DIX
Alexander, Fred — Capt. (M.C.), Station Hospital, ALR
Flanders, Merton Newcomb — Tilton General Hospital, ALR
Levbarg, John J. — Tilton General Hospital, ALR
Mountain, E. R. — Reception Center, ALR
McKee, Thomas L. — Maj. (M.C.), Tilton General Hospital, OP

GREYSTONE PARK
Gambill, Perry Judson — New Jersey State Hospital, ALR

FORT HANCOCK
Coleman, Joseph A. — Capt. (M.C.), Station Hospital, ALR
Foster, Clarence B. — Capt. (M.C.), 52nd Station Hospital, OP

HACKENSACK
Berke, Raynold N. — 430 Union, OP
Dezer, Charles N., Jr. — 210 Main, ALR
Greenfield, William John — 50 Anderson, ALR
Hensle, Otto S. — Maj. (M.C.), 100 Prospect av., ALR
Liva, Arcangelo — 5 Pangborn place, OP
Spiegelglass, A. B. — 417 Main, ALR

HADDONFIELD
Shafer, A. H. — Maj. (M.C.), E. Greenman road, ALR

JERSEY CITY
Pyle, Wallace — 15 Exchange place, OALR
Zbar, Joseph E. — Capt. (M.C.), 307 Varick st., ALR

LONG BRANCH
Campbell, W. K. — 96 Third av., OALR

MONTCLAIR
Agnew, Hobart McV. — 17 Plymouth, OP
Moore, James Alanson — Capt. (M.C.), 99 S. Mountain av., ALR
Shannon, James B. — 66 S. Fullerton av., ALR
Shannon, L. M. — 66 S. Fullerton av., ALR

MOORESTOWN
Ulmer, David H. B. — 199 Chestnut, ALR

MORRISTOWN
Byrne, J. Arthur — 16 Elm, ALR
Mathews, Raymond H. — 186 South, ALR

NEWARK
Adelman, B. B. — 190 Clinton av., OP
Eagleton, Wells P. — 15 Lombardy, ALR
Goldstein, Henry Z. — 31 Lincoln Park, ALR
Hahn, William H. — 15 Lombardy, OP
Hughes, L. W. — 965 Broad, OP
Hurff, J. Wallace — 86 Washington, OALR
Keim, William F. — 25 Roseville av., ALR
Krone, William Frank — 31 Lincoln Park, ALR
O'Connor, D. F. — 671 Broad, OALR
Orton, Henry B. — 224 Delavan av., ALR
Peer, L. A. — 965 Broad, ALR
Roh, Robert Franklin — 671 Broad, ALR
Sherman, A. Russell — 671 Broad, OP
Sherman, Elbert S. — 671 Broad, OP
Wood, Earl LeRoy — Col. (M.C.), 225 Ballantine Parkway, ALR
Zehnder, A. Charles — 188 Roseville av., OP

NEW JERSEY — Continued

NEW BRUNSWICK
Kler, Joseph Henry....151 Livingston av., ALR

ORANGE
Emerson, Linn....303 Park av., OP

PATERSON
Atwood, Edward A....360 Park av., ALR

PERTH AMBOY
Cohen, Nathan B....Maj. (M.C.), 104 Market, OALR
Mann, J. J....255 State, ALR

PLAINFIELD
Childers, Robert Jefferson....604 Park av., ALR

PRINCETON
Jellis, Walter Lt. Comdr. (M.C.), Princeton University, ALR

RIVERSIDE
Geary, Russell Diener....337 Bridgeboro road, ALR

SOUTH ORANGE
Miller, Joseph A....364 Prospect st., ALR

SUMMIT
Burritt, Norman W....30 Beechwood road, ALR
Sly, John L....382 Springfield av., ALR

TEANECK
Littwin, Charles....950 Queen Anne road, OALR

TRENTON
Ivins, William Clifford....455 W. State, ALR
Levy, Irvin....154 W. State, OP

UNION CITY
Conty, Anthony J....318 Forty-eighth, ALR
Pagliughi, John Joseph....401-18 Street, ALR
Sacco, Anthony G....2200 New York av., OALR

WESTFIELD
Tomlinson, Rolland Davis....445 E. Broad, ALR

WEST NEW YORK
Selinger, Samuel....413 Sixtieth, OALR

NEW MEXICO

ALBUQUERQUE
Brehmer, Harrison L....First Nat'l Bank bldg., OALR

ROSWELL
Griswold, George Wayne....211 W. 3rd, ALR

SANTA FE
Barton, William C....104½ W. San Francisco, OALR
Pumphrey, Gordon H.
....Maj. (M.C.), Bruns General Hospital, OP

NEW YORK

ALBANY
Bedell, Arthur J....344 State, OP
Dowling, J. Ivimey....116 Washington av., OALR
Freund, E. Martin....762 Madison av., ALR
Holohan, Joseph Leo....330 State, ALR
Judge, Harry V....46 Willett, OP
Kemp, Roy C....343 State, ALR
Mott, Walter Charles....Maj. (M.C.), 103 Lenox av., OP
Pratt, Emily Adelaide....State Education dept., ALR
Simon, Emanuel....577 Western av., ALR
Tebbutt, H. K....240 State, ALR
Volk, Benjamin M....146 Washington av., ALR

AUBURN
Bullard, C. W....120 Genesee, ALR
Wiley, Jason L....Metcalf bldg., OP
Wood, Everett H....120 Genesee, OP

BATAVIA
Bonner, Wm. F....Veterans Administration Facility, OALR
Wilson, Robert G....304 East Main st., ALR

BATH
Adamski, Michael S....8 Allen, OP

BAY SHORE
McGrath, Harry....12 Shore Lane, OP

BINGHAMTON
Levy, Harry H....119 Murray, OP
Monserrate, Manuel Maria....73 Main, ALR
Randall, Hiram....81 Main, ALR
Smith, Windsor R....67 Front, ALR
Vogt, George C....140 Chapin, ALR
Watson, H. de Witt....151 Front, OALR

BRONXVILLE
Carter, William W....6 Chestnut av., ALR
McKeown, Hugh S....9 Tanglewylde av., OP
Petruzzi, William Astor....65 Pondfield road, ALR
Ross, John....37 Oriole av., ALR

BROOKLYN
Agan, William B....1 Nevins, OP
Baker, John Percy....51 S. Oxford, ALR
Bakst, Max....186 New York av., ALR
Behrens, Alvin....9 Prospect Park West, OP
Beinfield, Henry H....760 Eastern Parkway, ALR
Bergmann, Milton B....959 Bushwick av., OP
Blankfield, Nathan....429 Lincoln pl., Station B, ALR
Blumenfeld, Louis....221 Brooklyn av., ALR
Brooks, Morris J....455 Ocean av., ALR
Bruno, Joseph M. L....31 Jefferson, OP
Buonaguro, Michael J....589 Lorimer, OP
Campbell, G. S.
Comdr. (M.C.), U. S. Naval Medical Supply Depot, ALR
Cutino, R. M....79 Pierrepont, OP
Cutler, Abraham Maurice....750 Ocean av., ALR
DeCoste, S. H....1179 Dean, ALR
Ebeling, William B....54 Greene av., OP
Evans, J. N....23 Schermerhorn, OP
Feldman, Joseph....668 Eastern Parkway, ALR
Feldman, Louis A....259 New York av., OP
Fine, Abraham....1505 Ocean av., OALR
Fisher, George Garthwaite....271 New York av., ALR
Freiman, George....99 Lafayette av., OP
Garofalo, Charles Joseph....439 Seventy-seventh, ALR
Gerzog, Bennett G....891 St. Marks av., ALR
Gilligan, Alexander....1154 Dean, ALR
Golden, Matthew G....1 Nevins, ALR
Golding, Joseph E....1 Nevins, OP
Gottlieb, Bernard N....387 Eastern Parkway, ALR
Greenfield, Samuel D....169 New York av., ALR
Griffin, Edwin A....18 Prospect Park, West, ALR
Gubner, Julius....25 Lefferts av., ALR
Hargitt, Charles A....17 Schermerhorn, OP
Harris, Morris L....20 Plaza st., ALR
Holzberger, John C....7005 Freshpond, ALR
Hull, Allen....401 76th st., OP
Kaplan, Carl....69-17 Forest av., ALR
Kasnetz, Joseph P....678 Eastern Parkway, ALR
Kaufman, Maxwell....85 Maujer, ALR
Kayser, Reuben....442 Bay Ridge Parkway, 75th st., ALR
Keil, Peter A....118 Pennsylvania av., ALR

NEW YORK — Continued

Kopp, Mortimer M. ..... 79 Argyle road, ALR
Kravitz, Daniel ..... 861 Park place, OP
Kreag, Karl A. ..... 167 Joralemon, ALR
Kruskal, I. D. ..... 101 Lafayette av., OP
Kuritzky, Isadore ..... 1480 Nostrand av., ALR
Landesberg, Jacques ..... 515 Ocean av., OP
Lloyd, Ralph I. ..... 14 Eighth av., OP
Lombardo, Melchiore ..... 142 Joralemon, OP
Mallon, Frank E. ..... 1135 Park place, OP
Menger, Louis Charles ..... 202 Weinfield, ALR
Mensher, Ira Wadsworth ..... 50 Plaza st., OP
Mezz, David ..... 354 New York av., ALR
Moehle, Walter ..... 15 Schermerhorn, OP
Moore, Walter V. ..... 1 Nevins, OP
Moorhead, Robert L. ..... 125 Remsen, ALR
Nelson, Nathaniel F. ..... 4910 15th av., OP
Neuberger, Julius Frederick .....
..... Comdr. (M.C.), U. S. Naval Hospital, ALR
Nova, Jules M. ..... 349 Adelphi, OP
O'Brien, G. R. ..... In Service, 307 Sterling place, ALR
Ohly, John H. ..... 22 Schermerhorn, OP
Place, E. Clifford ..... 59 Livingston, OP
Rabbiner, Max ..... 358 New York av., ALR
Rizzuti, A. Benedict ..... 160 Henry st., OP
Rosenthal, Benjamin Charles ..... 140 New York av., OP
Ruchman, Jacob ..... 301 Brooklyn av., ALR
Saskin, Edward ..... 855 St. Marks av., OP
Schmidt, James M. ..... 40 S. Oxford, ALR
Shapin, Sydney ..... 1346 Fiftieth, ALR
Sher, Maurice A. ..... 5022 Fifteenth av., ALR
Shorr, Abraham ..... 225 Eastern Parkway, ALR
Silberman, Meyer ..... 266 Brooklyn av., ALR
Silverberg, Joseph S. ..... 755 Ocean av., ALR
Smith, Louis S. ..... 2017 63rd, ALR
Sparber, Herman ..... 4701 Fifteenth av., OP
Steinbugler, William F. C. ..... 545 Ocean av., OP
Stern, Wilhelm E. ..... 1099 Bushwick av., ALR
Sunde, Einar A. ..... 627 Second, ALR
Thaler, Nathan ..... 570 Eastern Parkway, ALR
Tucker, Frank Landale, Jr. ..... 464 Ninth st., ALR
Usen, Louis ..... 278 New York av., ALR
Wechsler, A. H. ..... 340 New York av., ALR
Weeth, Charles R. ..... 37 Remsen, ALR
Weinstein, Samuel ..... 441 Ocean av., ALR
Wieselthier, Maurice L. ..... 1322 Union st., OP
Woodward, John F., Jr. .....
..... Lt. Comdr. (M.C.), Naval Hospital, ALR
Zwerling, Samuel ..... 789 St. Mark's av., ALR

BUFFALO

Beyer, Louis J. ..... 449 Delaware av., ALR
Blauuw, E. E. ..... 190 Ashland av., OP
Bozer, Herrmann Eugene ..... 468 Delaware av., ALR
Brown, C. M. ..... 510 Delaware av., ALR
Buffum, Edward S. ..... 40 W. North, ALR
Cott, C. C. ..... 333 Linwood av., ALR
Cowper, H. W. ..... 543 Franklin, OP
Delman, A. H., Maj. (M.C.),
U.S.A. Induction Station, Old Customs House, ALR
Fairbairn, John F. ..... 925 Delaware av., ALR
Fowler, James Gwynne ..... 412 Linwood av., OP
Freeman, Sheldon B. ..... 196 Linwood av., OP
Frost, Carl Grover ..... 333 Linwood av., ALR
Gerstner, Martin L. ..... 468 Delaware av., ALR
Glosser, Herbert H. ..... 448 Franklin, OP
Hague, Elliott B. ..... 454 Franklin, OP
Harnick, Israel ..... 1132 Delaware av., ALR
Howard, Wm. M. ..... 389 Linwood av., OP
King, Walter F. ..... 519 Delaware av., OP
Koenig, Ivan J. ..... 40 W. North, OP
Krystaf, Joseph George ..... 65 Vernon place, ALR
Le Win, Thurber ..... 112 Linwood av., OP
Luhr, A. F. ..... 40 W. North, OP
McGregor, d'Arcy ..... 83 Bryant, ALR
March, Clara A. ..... 465 Ashland, OP
Palmer, Milton A. ..... 2186 Seneca, OP
Rosenberg, Joseph ..... 333 Linwood av., ALR
Scanio, Joseph Charles ..... 333 Linwood av., ALR
Schutz, Joseph A. ..... 2804 Main, OP
Smith, Leon ..... 467 Linwood av., ALR
Trotter, Homer A. ..... 40 W. North, ALR
Wurtz, W. J. M. ..... 333 Linwood av., ALR

CORTLAND

Higgins, R. P. ..... 20 Court, OALR

EAST AURORA

Andrews, H. D. ..... 429 Oakland av., OP

ELMIRA

Boland, William Tilden ..... 315 Lake, OP
Glover, Arthur C. ..... 311 W. Church, OALR

FAR ROCKAWAY

Hirschmann, Hans A. ..... 1502 Mott av., ALR

FLUSHING

Goode, Edward Allen ..... 143-28 Sanford av., ALR
Weiss, Harry ..... Capt. (M.C.), 140-50 Ash av., OP

FORT TOTTEN

Vadala, Anthony J. ..... Lt. Col. (M.C.), Station Hospital, OALR

FREEPORT, L. I.

Rossman, Morris Muncie .....
..... Lt. (M.C.) (j. g.), U.S.N.R., 115 N. Main, ALR

GARDEN CITY

Wood, Christopher ..... 87 7th st., OP

GASPORT

Pierce, Wm. W. ..... Capt. (M.C.), ALR

GENEVA

Spengler, John Arthur ..... 423 S. Main, OP

GLENS FALLS

Buck, Clair A. ..... Maj. (M.C.), 191 Glen, ALR

HEMPSTEAD

Austin, M. F. ..... Maj. (M.C.), 231 Main st., ALR
Coon, Eugene H. ..... 131 Fulton av., ALR
Hughes, Wendell L. ..... Professional bldg., OP
Smith, Henry Buel ..... 131 Fulton av., ALR

HOLLIS

Bunnell, C. W. ..... Lt. Comdr. (M.C.), 9115 193rd st., ALR
Roemaet, Charles M. ..... 191-01 Hollis av., ALR

HUNTINGTON

Kimbrig, Max ..... 20 High, ALR

ITHACA

Wilson, Hudson J. ..... First Nat'l Bank bldg., ALR

JAMAICA

Bender, M. S. ..... 88-05 150th, ALR
Douglas, Edward M. ..... 87-85 153rd, OP
Kane, J. T. ..... 88-49 163rd, ALR
Keeler, Lansing H. ..... 89-04 148th, OALR
Miller, Saul ..... 88-05 150th, OP

NEW YORK — Continued

JAMESTOWN
Bourne, Malcolm G. ... 110 W. 3rd, OP

JOHNSON CITY
Hudock, John Paul ... 67 Broad st., ALR

JOHNSTOWN
Sarno, J. Frederick ... 109 S. William st., ALR

KINGSTON
Wilson, Harold A. ... 71 Maiden Lane, ALR

LANCASTER
Palmer, Milton Alwood ... 18 Park blvd., OP

LAURELTON
Landon, H. Robert ... Laurelton Professional bldg., ALR

LITTLE FALLS
Newton, Morris H. ... Burrell bldg., OP

LOCKPORT
Morgana, Dante James ... 13 Main, OP
Parker, Harry L. ... 88 East av., ALR

LONG ISLAND CITY
Gewanter, Robert ... 41-44 44th st., ALR
Hulka, J. H. ... 30-38 36th st., OALR

LYNBROOK
Gerstley, Manfred J. ... In Service (address unknown), ALR
Hagan, Cornelius E., Jr. ... Capt. (M.C.), 31 Evergreen av., ALR

MIDDLETOWN
Hicks, William J. ... 15 King, ALR
Neller, Walter I. ... 121 Wickham av., OP

MILLERTON
Tabor, Gilbert Stevens ... 11 Park Row, ALR

MT. VERNON
Houlahan, James M. ... 117 S. Second av., OP
Keefe, William John ... 205 E. Sidney av., OALR
Yavelow, Charles S. ... 244 Bedford av., ALR

NEWBURGH
Browning, Birt Lee ... 208 Grand, ALR
McWilliam, Charles S. ... 205 Liberty, OP

NEW ROCHELLE
Beck, A. L. ... 421 Huguenot, OALR
Edmunds, Stanley E. ... Lt. (M.C.), U.S.N.R., 86 Perth av., ALR
Hetrick, L. E. ... 260 North av., ALR
Smith, Morley T. ... 421 Huguenot, ALR
Somberg, Jos. S. ... 524 North av., OP

NEW YORK CITY
Aebli, Rudolph ... 30 E. 40th, OP
Allan, William B. ... 410 Park av., OALR
Almour, Ralph ... In Service (address unknown), ALR
Amsel, Maxwell R. ... 145 W. 55th, ALR
Anderson, Orrin E. ... Capt. (M.C.), 39 Whitehall st., ALR
Atkinson, Miles ... 123 E. 61st, ALR
Auerbach, Julius ... 897 Park av., ALR
Austen, Willard Emerson ... Lt. Comdr. (M.C.), 115 E. 61st st., ALR
Baker, Daniel C. ... 180 Fort Washington av., ALR
Ballin, M. J. ... 77 Park av., ALR
Barnes, Mark Hopkins ... 324 E. 41st st., OALR
Bassen, Edward J. ... Maj. (M.C.), 654 Madison av., OP
Bassett, David I. ... 1075 Grand Concourse, ALR
Beale, A. W. ... A.P.O. 886, c/o Postmaster, ALR
Bell, Julius W. ... 200 E. 16th, ALR
Bender, M. S. ... 121 E. 60th, ALR
Berens, Conrad ... 35 E. 70th, OP
Berger, Isidore ... 888 Grand Concourse, Bronx, ALR
Berger, Murray ... 300 Central Park West, ALR
Berliner, M. L. ... 57 W. 57th, OP
Bilchick, Edwin B. ... Maj. (M.C.), 216 W. 89th, ALR
Bogart, Donald Weeks ... 20 E. 53rd, OP
Bonaccolto, Girolamo ... 654 Madison av., OP
Bowers, W. C. ... 17 E. 61st, ALR
Boyce, T. L. ... 654 Madison av., OP
Braley, Alson ... 630 W. 168th, OP
Branigan, C. Hugh ... 114 E. 54th st., OP
Breuning, Paul H. ... 133 E. 58th, ALR
Brighton, George R. ... 580 Park av., ALR
Broones, Otto ... 302 W. 87th, ALR
Bruce, G. M. ... Lt. Comdr. (M.C.), 635 W. 165th, OP
Bruder, Joseph ... 57 W. 57th, OALR
Burman, Herman J. ... 1235 Grand Concourse, ALR
Carroll, Frank D. ... Eye Institute, 635 W. 165th, OP
Castroviejo, Ramon ... 635 W. 165th, OP
Cinelli, Albert A. ... 101 E. 85th, ALR
Cohen, Martin ... 29 E. 64th, OP
Coleman, Lester Laudy ... 162 E. 80th, ALR
Conboy, John E. ... 2265 University av., OP
Coryn, Harold W. ... 30 E. 40th, ALR
Creasy, Raymond C. ... 67 E. 59th, ALR
Crespo, Jose R. ... 150 W. 87th, ALR
Cunning, Daniel S. ... 121 E. 60th, OALR
Cutler, Harold M., Capt. (M.C.), 21st Station Hospital, A.P.O. 685, c/o Postmaster, ALR
Daley, Jacob ... 104 E. 40th, ALR
Davidson, Morris ... 80 Centre, OP
Davis, G. E. ... 1060 Fifth av., OALR
Davis, Hugh Peter, Maj. (M.C.), 9th Evacuation Hospital, A.P.O. 302, c/o Postmaster, ALR
Delaney, Adrian J., Lt. Comdr. (M.C.), U.S.S. Boston, c/o Postmaster, ALR
Denison, Ward C. ... Lt. Comdr. (M.C.), Navy 602, c/o Fleet P. O., ALR
Depping, Charles William ... 17 W. 54th, ALR
De Voe, Gerard ... 30 W. 59th, OP
Dewey, C. H. ... 90 Church st., OALR
Dillemuth, Frederick Joseph ... 404 E. 154th, ALR
Dixon, G. S. ... 255 W. 108th, R
Dlugasch, Louis ... 110 E. 36th, ALR
Doherty, W. B. ... 150 W. 55th, OP
Druss, Joseph George ... 1040 Park av., ALR
Dunnington, J. H. ... 30 W. 59th, OP
Dutrow, H. V. Maj. (M.C.), A.P.O. 845, c/o Postmaster, OALR
Dwyer, James G. ... 375 Park av., ALR
Ellis, Z. H. ... 58 E. 65th OP
Esterman, Benjamin ... 983 Park av., , OP
Everett, A. Reginald ... 33 E. 68th st., ALR
Falkson, Kurt F. ... 229 W. 78th st., ALR
Farrior, J. B. ... Capt. (M.C.), 298th General Hospital, A.P.O. 508, ALR
Fernlund, George Byron ... 8620 115th st., Richmond Hill, ALR
Fishof, Frank E. ... 860 Grand Concourse, ALR
Fitzgerald, Fred J. C. ... 118 E. 65th, OP
Flack, J. Vincent ... Lt. Comdr. (M.C.), 90 Church, OP
Foster, Harold ... 92-11 35th av., Jackson Heights, ALR
Fowler, Edmund Prince, Jr. ... 180 Ft. Washington av., ALR
Fowlkes, John Winston ... 12 E. 63rd, ALR
Fox, Sidney Albert ... Maj. (M.C.), 138 E. 36th st., OP
Francis, Charles C. ... 610 West End av., ALR

NEW YORK — Continued

Frank, David I., Capt. (M.C.), 10th Field Hospital, A.P.O. 3658, c/o Postmaster, ALR
Frazee, John R.....6th General Hospital, A.P.O. 668, ALR
Fread, Bernard.....45 E. 85th, OP
Frey, W. G., Jr.....121 E. 60th, OP
Friedman, Benjamin.....6 W. 77th, OP
Friesner, Isidore.....101 E. 73rd, A
Frothingham, Florie D.....58 E. 58th, ALR
Furman, Martin A.....Maj. (M.C.), 3875 Waldo av., ALR
Garrick, Thomas J.....40 E. 61st, ALR
Gartner, Samuel.....1749 Grand Concourse, OP
Gatewood, Wm. L.....111 E. 61st st., ALR
Gilmore, Goodlatte Browne.....2940 Grand Concourse, ALR
Givner, Isadore.....108 E. 66th, OP
Glushak, Leopold.....1 West 85th, ALR
Goldman, H. G.....57 W. 58th, ALR
Goldman, Irving Brice.....121 E. 60th, ALR
Gould, Nathaniel Jay.....940 Grand Concourse, OP
Graebner, Herbert.....45 Park av., ALR
Graef, Fred W.....850 Park av., ALR
Grant, Maurice David.....1860 Morris av., ALR
Greene, James S.....61 Irving place, ALR
Greenfield, William J.....777 West End av., ALR
Gresser, Edward Bellamy.....39 E. 75th, OP
Griesman, Bruno L.....1100 Park av., ALR
Griffey, Walter P.....Marine Hospital, Stapleton, OP
Griffith, Charles M.....Comdr. (M.C.), 140 E. 54th st., ALR
Grimaldi, Americo John.....100 W. 59th, OP
Grove, Russell Clark.....60 E. 58th, ALR
Grushlaw, Israel.....225 W. 86th, ALR
Gurtov, John Jacob.....1235 Grand Concourse, ALR
Guy, Loren P.....40 E. 62nd, OP
Hanley, James Swift.....40 W. 55th, ALR
Hardy, LeGrand H.....23 E. 79th, OP
Hartshorne, Isaac.....30 W. 59th, OP
Haskin, W. H.....375 Park av., ALR
Herzig, Arthur J.....667 Madison av., ALR
Hetrick, J. A. Werner.....667 Madison av., ALR
Hetrick, L. E.....40 E. 61st, ALR
Holden, William H.....3 E. 85th, ALR
Hory, Joseph S.....2488 Grand Concourse, OP
Hunt Westley Marshall.....907 5th av., ALR
Huvelle, Rene H.....30 E. 40th, ALR
Hynes, Frank Joseph.....121 E. 60th, ALR
Ide, David.....1749 Grand Concourse, ALR
Imperatori, C. J.....108 E. 38th, ALR
Ingerman, S. M.....418 Central Park West, OALR
Insel, Herman Herbert.....Welfare Island, ALR
Jacobius, Lawrence.....2021 Grand Concourse, ALR
Jacobs, S. M.....1942 Grand Concourse, OALR
Jaffe, Morris.....940 Grand Concourse, OP
Jessel, Erwin.. Lt(M.C.), 1749 Grand Concourse, Bronx, OP
Johnson, Thomas Holland.....30 W. 59th, OP
Jones, David H.....140 E. 54th, LR
Jones, Marvin F.....121 E. 60th, ALR
Judd, H. B.....40 E. 61st, ALR
Kafka, M. Martyn.....67 E. 59th, ALR
Keil, Frank C.....660 Madison av., OALR
Kelly, Joseph Dominic.....140 E. 54th, OALR
Kend, Lee.....133 E. 58th, ALR
Kirby, D. B.....780 Park av., OP
Kirkbright, James C.....40 E. 61st, ALR
Kleinfeld, Louis.....17 E. 84th, ALR
Knapp, Arnold.....10 E. 54th, OP
Knighton, W. S.....121 E. 61st, OP
Knopf, Saul.....30 E. 40th, ALR
Kolfer, Carl.....68 E. 86th, OP
Kopetzky, S. J.....71 E. 80th, ALR
Kornberg, Maurice.....441 E. 15th, ALR
Kornzweig, Abraham L.....1200 Fifth av., OP
Kronenberg, Bernard.....162 W. 54th, OP
Krug, Ernest F.....988 Fifth av., OP
LaGattuta, Frank.....3754 White Plains av., Bronx, OP
Lambert, Robert K.....1010 Fifth av., OP
Last, Murray A.....1123 Park av., OP
Laval, Joseph.....136 E. 64th, OP
La Vigne, A. A.....667 Madison av., OALR
Law, Frederick M.....140 E. 54th, ALR
Lefkon, Irving M.....37-14 73rd, Jackson Heights, ALR
Liggett, Harold.....133 E. 58th, ALR
Limbach, Earl Frederick.....30 W. 59th, Station "G", ALR
Loewe, Walter R., Comdr. (M.C.), U .S. Navy, In Service (address unknown), OP
Lore, John M.....325 West End av., ALR
Lubman, Max.....50 Manhattan av., ALR
Lupo C. W., Col. (M.C.), 113th Station Hospital, A.P.O. 3553, c/o Postmaster, ALR
McAuliffe, Gervais W.....110 W. 55th, ALR
McClanahan, Robert.....60 E. 75th, OP
McCuaig, Duncan Roderick, Maj. (M.C.), O-413966, 2nd Evacuation Hosp., A.P.O. 511, c/o Postmaster, ALR
McCuaig, Victor C.....40 E. 61st, ALR
McDannald, C. E.....100 W. 59th, OP
McGavic, John Samuel.....635 W. 165th, OP
McGrath, James B.....2615 Grand Concourse, ALR
McKeown, Hugh S.....635 W. 165th, OP
McLean, John M.....525 E. 68th, OP
Macdonald, James O.....67 E. 59th st., ALR
MacDonald, Philip G.....115 E. 61st, ALR
Macpherson, Duncan.....920 Fifth av., ALR
Maliniac, Jacques W.....106 E. 78th, ALR
Martin, Ashby G.....995 Lexington av., ALR
Matusow, Abraham P.....1100 Grand Concourse, ALR
May, Charles H.....70 E. 66th, OP
Maybaum, J. L.....1070 Park av., ALR
Miller, Isidore.....2021 Grand Concourse, ALR
Miller, Joseph William.....242 E. 19th, ALR
Minsky, Henry.....2 E. 95th, OP
Mirow, David.....237 E. 20th, ALR
Mittendorf, A. D.....399 Park av., OP
Moghtader, Mansoor.....40 E. 62nd, ALR
Morrison, W. W.....Lt. Comdr. (M.C.), 320 E. 42nd, ALR
Moshinsky, Alexander L.....237 E. 20th, ALR
Moyer, John S.....410 Park av., ALR
Myerson, M. C.....136 E. 64th, ALR
Neer, Edmonde DeWitte.....115 E. 61st, ALR
Neuschatz, Gerald.....327 Central Park West, ALR
Nisselson, Max.....250 Riverside drive, ALR
Northcott, Thomas A.....40 E .61st, OP
Northington, Page.....In Service, 77 Park av., ALR
Opin, Edward.....2065 Grand Concourse, ALR
Page, John R.....127 E. 62nd, ALR
Palmer, Arthur.....667 Madison av., ALR
Paton, R. T.....927 Park av., OP
Payne, B. F.....In Service, 896 Madison av., OP
Pearlman, Louis M.....53 E. 93rd, ALR
Perera, Charles A.....70 E. 66th, OP
Perkins, Osborn P.....121 E. 60th, OP
Pfeiffer, Raymond L.....635 W. 165th, OP
Pullen, Edward Markey.....30 E. 40th, ALR
Reese, Algernon B.....73 E. 71st, OP
Risch, Otto C.....104 E. 40th, ALR
Rolett, D. M.....876 Park av., OP
Rosen, Sam.....101 E. 73rd, ALR
Rosenbaum, Morris.....61 W. 88th, OP

NEW YORK — Continued

Rosenbluth, Michael.....225 W. 106th, ALR
Rosenwasser, Harry.....101 E. 73rd st., ALR
Ross, John.....6 E. 70th, ALR
Rothberg, Maurice.....
.....Maj. (M.C.), A.P.O. 862, c/o Postmaster, OP
Rubenstein, Hyman John.....510 Madison av., ALR
Ryan, Maxwell D.....660 Madison av., ALR
Sackin, David.....“St. Morits,” 50 Central Park South, ALR
Saltzman, Samuel L.....334 W. 86th, OP
Samuels, Bernard.....57 W. 57th, OP
Saunders, Truman L.....103 E. 65th, ALR
Seal, Joseph Coleman.....10 E. 96th, ALR
Schechter, Solomon.....1749 Grand Concourse, ALR
Scheer, Henry M.....522 West End av., ALR
Scheie, H. G., Capt. (M.C.), 20th General Hospital,
A.P.O. 3492, c/o Postmaster, OP
Schillinger, Raphael.....390 West End av., ALR
Schlivek, Kaufman.....1016 Fifth av., OP
Schmidt, Otto V. M.....568 Park av., ALR
Schoenberg, Mark J.....667 Madison av., OP
Schugt, Henry P.....122 E. 76th, ALR
Schwartz, A. A.....7 W. 71st, ALR
Schwartz, Harry.....1749 Grand Concourse, OP
Schwartz, Leo.....136 E. 57th, ALR
Seligman, Albert E.....41 Forest av., Staten Island, ALR
Seward, J. A.....115 E. 61st, ALR
Sitchevska, Olga L.....30 Fifth av., OP
Smith, Clarence Henry.....140 E. 54th, ALR
Smith, James M.....123 E. 53rd, ALR
Smith, James W.....1016 Fifth av., OP
Snyder, Eugene Ralph.....1100 Park av., ALR
Somberg, J. S.....20 E. 87th, OP
Sparer, William S.....50 E. 10th, ALR
Stark, Jessie B.....46 Park av., OP
Steinbach, Louis.....1882 Grand Concourse, OP
Stone, L. O., Capt. (M.C.), U.S.N.,
U. S. Naval Air Station, Bermuda, c/o Postmaster, ALR
Stovin, Joseph S.....151 Central Park West, ALR
Straatsma, Clarence R. Lt. Comdr. (M.C.), 66 E. 79th, ALR
Street, Alvin Murray.....745 5th av, ALR
Strickler, Joseph George.....200 W. 57th, ALR
Sturges, Leigh F.....121 E. 60th, ALR
Swanson, Clifford A., Comdr. (M.C.), U.S.S. Iowa,
c/o Fleet Postmaster, ALR
Swick, David A.....30 W. 70th, OP
Syracuse, Victor R.....29 Washington Sq. W., OP
Taterka, Henry David.....302 W. 87th, ALR
Tickle, Thomas G.....107 E. 63rd, ALR
Troncoso, Manuel U.....500 West End av., OP
Tusak, E. A.....115 E. 90th, OP
Vogel, Felix P. S.....1018 E. 163rd, ALR
Voislawsky, Van Rensselaer.....33 E. 68th, ALR
Watnick, Joseph Louis.....10 W. 96th, ALR
Webster, David H.....140 E. 54th, OP
Weil, Sidney.....1840 Grand Concourse, OP
Wexler, David.....103 E. 86th, OP
Weymuller, E. A.....135 E. 64th, ALR
Wheeler, Maynard C.....30 W. 59th, OP
White, Francis W.....471 Park av., ALR
White, James W.....15 Park av., OP
Wiener, Alfred.....550 Park av., OALR
Wincor, Henry G.....1882 Grand Concourse, OP
Woodman, DeGraaf.....156 E. 37th, ALR
Zuckerman, Joshua.....108 E. 91st, OP

NIAGARA FALLS

Goetzman, Arthur Cecil.....8907 Hennepin av., OP
Price, Norman W.....202 Gluck bldg., OALR
Rooker, A. M.....United bldg., ALR
Walker, Oliver Frank.....2873 Lewiston road, ALR

NYACK

Theis, Franklyn B.....209 S. Broadway, ALR
Toms, S. W. S.....120 S. Broadway, OALR

OLEAN

Moore, Ellis Columbus.....First Nat'l Bank bldg., ALR

ONEIDA

Crockett, R. L.....302 Main st., OP

ONEONTA

Hall, Edwin P.....76 Chestnut, ALR

ORANGEBURG

Wegner, E. A., Jr.....
.....Capt. (M.C.), Station Hospital, Camp Shanks, ALR

OSSINING

Newlands, William A.....16 Maple place, ALR

OSWEGO

Masor, Philip L.....
.....Lt. (M.C.), Station Hospital, Ft. Ontario, OP

POUGHKEEPSIE

Krieger, William A.....103 Hooker av., OALR
Voorhees, Earle W.....8 Garfield place, ALR

QUEENS VILLAGE

Pauley, Gerald E.....9460—220th st., ALR

ROCHESTER

Avery, Elroy J.....11 N. Goodman, ALR
Barber, F.....75 S. Fitzhugh, OP
Bishop, Vernon L.....277 Alexander, ALR
Clark, L. H.....337 Monroe av., OALR
Elliott, Raymond E.....76 S. Fitzhugh, ALR
Fischer, Abraham.....Mercantile bldg., OP
Gipner, J. F.....Medical Arts bldg., OP
Goldstein, Harry Z.....Lt. (M.C.), Induction Center, ALR
Gorin, Meyer.....35 Chestnut st., ALR
Hawkins, Raymond W.....39 S. Goodman, ALR
Hilleman, George A.....267 Alexander, ALR
Honiss, John Ralph.....277 Alexander, ALR
Jones, Leonard W.....16 N. Goodman, OALR
Kennedy, Eldred Weston.....Gas and Electric bldg., OP
Lerner, Macy Levi.....332 Park av., OP
McDowell, N. D.....275 Alexander, OALR
Morris, A. G.....Medical Arts bldg., ALR
Nash, C. Stewart.....277 Alexander, ALR
O'Brien, Elmer W.....5 N. Goodman, ALR
Shapero, I. M.....259 Alexander, OALR
Snell, Albert C.....53 S. Fitzhugh, OP
Sullivan, Charles T.....277 Alexander, OP
Trainor, George McCague.....
.....Capt. (M.C.), 195 Wilshire road, ALR

ROCKVILLE CENTER, L. I.

Goldman, A. Milton.....1 N. Village av., OP

ROME

Valone, Frank H.....107 W. Court, OP

RYE

Carroll, Frank D.....Blind Brook Lodge, OP
Peterson, N. Vern.....Highland Hall, ALR

ST. ALBANS

Fackler, C. L., Lt. Comdr. (M.C.), Naval Hospital, L. I., OALR

SAMPSON

McLaughlin John J.....
.....Lt. (M.C.), U. S. Naval Training Station, ALR

NEW YORK — Continued

SARANAC LAKE
Beck, Robert J. ..... 105 Main, OP
Wilson, George E. ..... 108 Main, ALR

SARATOGA SPRINGS
Goodfellow, Thomas J. ..... Arcade bldg., OALR

SCARSDALE
Ryan, E. P. ..... Lt. Comdr. (M.C.), 18 Fairview road, OP

SCHENECTADY
Gulick, John Duncan ..... 704 Union, ALR
Ostrom, William C. ..... 1350 Union, OP

STATEN ISLAND
Conway, William S. .....
..... Capt. (M.C.), Halloran General Hospital, OALR
Kahn, K. M. ... Maj. (M.C.), Halloran General Hospital, ALR

SYRACUSE
Blaisdell, Irl Hazard ..... Medical Arts bldg., ALR
Brown, Mortimer G. ..... State Tower bldg., ALR
Brust, H. O. ..... 820 S. Crouse av., ALR
Cahill, James F. ..... 428 S. Salina, OP
Cave, Linus S. ..... State Tower bldg., ALR
Dickinson, William G. ..... State Tower bldg., OP
Gillette, D. F. ..... State Tower bldg., OP
Hoople, Gordon D. ... Maj. (M.C.), Medical Arts bldg., ALR
Joy, Harold Henry ..... State Tower bldg., OP
Katz, I. Herbert ..... 713 E. Genesee, OP
Kauffman, A. B. ..... Chimes bldg., ALR
Kline, H. G. ..... State Tower bldg., ALR
McClintock, Walter L. ..... 616 Oak, ALR
Moore, Roy S. ..... State Tower bldg., ALR
O'Connor, Frederick J. ..... State Tower bldg., ALR
Rubenstein, A. H. ..... Medical Arts bldg., ALR
Webster, F. R. ..... State Tower bldg., OP

TARRYTOWN
Newlands, W. Alexander ..... 19 S. Broadway, ALR

TROY
Sulzman, Frank Malcolm ..... 1831 Fifth av., OALR
Sulzman, John H. Lt. (M.C.), U.S.N.R., 1831 Fifth av., OP

UTICA
Beattie, William H. ..... 258 Genesee, OALR
Farrell, James I. ..... Lt. Comdr. (M.C.), 250 Genesee, OP
Jones, Fred G. ..... 258 Genesee, ALR

WARSAW
Nairn, George Waverly .....
..... Wyoming County Community Hospital, ALR

WATERTOWN
Atkinson, John H. ..... 168-70 Sterling, ALR
Atkinson, Walter S. ..... 129 Clinton, OP
Henderson, L. E. ..... 129 Clinton, OP
McAskill, James E. ..... Woolworth bldg., ALR

WEST POINT
Martin, H. F. ..... Maj. (M.C.), Station Hospital, ALR

WHITE PLAINS
Emery, John R. ..... Medical Centre, ALR
Knight, Granville F. ..... 900 North st., ALR
Moriarty, Richard W. ..... 3 Mamaroneck road, OALR

WOODHAVEN
Frothingham, Edward ..... 8526—94th, OALR
Stack, James Edward ..... 8858 Woodhaven blvd., ALR

YONKERS
Fitzgerald, Wm. W. ..... 27 Ludlow, OP
Petti, George Hyacinth ..... 4 Berkeley av., ALR
Wolcott, Charles C. ..... 27 Ludlow, ALR

NORTH CAROLINA

ASHEVILLE
Swann, Cecil C. ..... Flat Iron bldg., ALR
Weizenblatt, Sprinza ..... 709 New Medical bldg., OP

CHAPEL HILL
Combs, Arnold B. .....
..... Lt. (M.C.), U. S. Navy Preflight School, ALR

CHARLOTTE
Johnston, James G. ..... 1520 E. 4th, OP
Motley, Fred E. ..... 106 W. 7th, ALR
Neblett, Herbert Clarence ..... 401 N. Tryon, OP
Peeler, C. N. ..... 106 W. 7th, OALR
Schlessel, Louis ..... Maj. (M.C.), Morris Hill, ALR
Sloan, Henry L. ..... 106 W. 7th, OP
Smith, Franklin C. .....
..... Charlotte Eye, Ear & Throat Hospital, OP
Whisnant, A. M. ..... Professional bldg., OALR

DURHAM
Anderson, William Banks ..... Duke Hospital, OP
Eagle, Watt Weems ..... Duke Hospital, ALR
Ferguson, George Burton ..... 1114 West Main, ALR
Kerns, Thomas C. ..... 1110 W. Main, ALR
McPherson, Samuel D. ..... McPherson Hospital, ALR

FORT BRAGG
Arnold, Ralph A. .. Capt. (M.C.), 65th General Hospital, ALR

GOLDSBORO
Sheppard, Louis B. .....
..... Maj. (M.C.), Seymour Johnson Field, OP

GREENSBORO
Reaves, W. P. ..... 336 Church st., OALR
Taylor, Shahane R. ..... Jefferson bldg., ALR
Wolfe, Hugh C. ..... 102 N. Elm, ALR

HENDERSON
White, Clarence Hunt ..... Box 257, ALR

KINSTON
Peery, Vance Price ..... Kinston Clinic, ALR

MAXTON
Myers, David Capt. (M.C.), Station Hospital,
Laurinburg-Maxton, A.A.B., ALR

MORGANTON
Billings, Gilbert M. ..... 110 S. Sterling, ALR

RALEIGH
Hicks, Vennie Monroe ..... Professional bldg., OP
Powers, Frank P. ..... Professional bldg., ALR

SALISBURY
McCutchan, Frank ..... 100 W. Innes, ALR

SWANNANOA
Lathbury, Edwin .....
..... Capt. (M.C.), Moore General Hospital, ALR

WINSTON-SALEM
Bradford, George Edwin ..... Nissen bldg., ALR
Combs, Fielding ..... Nissen bldg., ALR
Davis, Thomas W. ..... O'Hanlon bldg., OALR
Harrill, James Albert ..... Reynolds bldg., ALR
Sink, Vergil Rex ..... 852 W. 5th, ALR
Speas, William Paul ..... Reynolds bldg., OP

NORTH DAKOTA

BISMARCK

Buckingham, Tracy W. — 405 Broadway, ALR
Constans, George Maurice — Clinic bldg., OP
Diven, Wilbur Lester — 402½ Main, ALR
Rosenberger, Henry P. — 221 Fifth, ALR

FARGO

Foster, George C. — 807 Broadway, OALR
Larson, G. Arthur — A.O.U.W. bldg., OALR
Rindlaub, Elizabeth P. — 321 Eighth South, OALR
Rindlaub, John H. — 321 Eighth South, OALR
Winn, William R. — Dakota Clinic, ALR

GRAND FORKS

Alger, L. James — Box 328, ALR
Jensen, August Frithiof — 322 De Mers av., OP
Youngs, Nelson A. — Grand Forks Clinic, ALR

MINOT

Brunner, George Harmon — McCannel Clinic bldg., OP
Lampert, M. T. — Northwest Clinic, ALR
McCannel, Archie D. — Corner 1st st., S.W. and Central av., OALR

OHIO

AKRON

Arbuckle, David S. — Akron Savings & Loan bldg., OALR
Brumbaugh, John D. — Second Nat'l Bank bldg., OALR
Mathias, Daniel Francis — Central Tower bldg., ALR
Parry, Thayer L. — Second Nat'l Bank bldg., OALR
Springer, James Earl — Peoples Bank bldg., ALR

ASHTABULA

Grimmett, Roger Skelton — 217 Park place, ALR
O'Connor, Timothy Francis — 4634 Main av., ALR

BELLEFONTAINE

Butler, Robert H. — 112 W. Columbus av., OALR
Harbert, John P. — 135 N. Main, OALR

CANTON

Hoffmann, John N. — Brant bldg., ALR
King, Clair Boyd — First Nat'l Bank bldg., OP
Rosedale, Raymond Samuel — 610 N. Market av., ALR
Schirack, Clarence J. — 817 Cleveland av. N.W., ALR
Toot, Joseph F. — First Nat'l Bank bldg., OP
Underwood, John H. — Brant bldg., ALR
Weaver, Homer V. — 625 Cleveland av. N.W., ALR

CHILLICOTHE

Shepard, Emerson P. — 72 West Second, ALR

CINCINNATI

Asbury, Mary K. — 3549 Holly Lane, OP
Beyer, A. G. — Carew Tower, OALR
Brown, Albert Louis — Carew Tower, OP
Finney, Nancy E. — 2525 Gilbert av., OP
Fischbach, Victor W. — Union Central bldg., ALR
Goodyear, H. M. — Doctors bldg., ALR
Haggart, Harry H. — Carew Tower, ALR
Hans, C. L. — Provident Bank bldg., OALR
Harris, W. C. — 19 W. 8th, ALR
Heidelman, George — Union Central bldg., OP
Hendricks, Louis J. — Carew Tower, OP
Hickman, W. R. — Lt. (M.C.), 424 E. 4th st., ALR
Hofling, Charles A. — 707 Race, OP
Hofmann, Albert P. — 630 Vine, ALR
Howard, Robert E. — Union Central bldg., ALR
Hyman, Merris — Doctors bldg., ALR
Iglauer, Samuel — 707 Race, ALR
Jones, C. C. — 19 Garfield place, ALR
King, Edward — Carew Tower, ALR
Levin, Abram Ascher — Carew Tower, OP
Lyle, Donald J. — Carew Tower, OP
Miller, Ralph H. — 441 Vine, OP
Mithoefer, W. — Carew Tower, ALR
Ray, Victor, Jr. — Carew Tower, OP
Reid, H. W. — Union Central bldg., OP
Safer, Louis A. — Provident Bank bldg., ALR
Sakler, Barnet R. — Capt. (M.C.), 695 N. Crescent, OP
Schrimpf, Cyril Edward — Union Central bldg., OP
Seltz, Samuel — Doctors bldg., ALR
Siegel, Francis X. — Union Central bldg., OALR
Stanbery, Henry — Doctors bldg., OP
Stewart, T. M. — Union Central bldg., OP
Stitt, H. L. — Doctors bldg., ALR
Stoll, Karl Ludwig — 19 W. 7th, OP
Swing, Frank U. — Doctors bldg., ALR
Tangeman, H. F. — Union Central bldg., ALR
Thompson, E. H. — 19 W. 7th, OALR
Vail, D. T. — Lt. Col. (M.C.), Carew Tower, OP
Woodward, Paul Maxwell — 4140 Hamilton av., ALR

CLEVELAND

Adelstein, Fred — Rose bldg., ALR
Allen, Donald Gray — 1021 Prospect av., OP
Brandwan, Samuel R. — 1851 E. 6th st., OP
Bruner, Abram Breneman — Guardian bldg., OP
Bruner, William E. — Guardian bldg., OP
Bucher, Walter M. — 4187 Pearl road, ALR
Burke, T. A. — Rose bldg., OALR
Chamberlain, Webb Parks — 7405 Detroit av., OP
Collins, James T. — Republic bldg., ALR
Deeton, William L. — 13125 Shaker square, ALR
Dixon, Fred Willis — Rose bldg., ALR
Engler, Clarence William — 2323 Prospect av., ALR
Forsythe, S. T. — Rose bldg., OALR
Garrett, Ernest W. — Hanna bldg., ALR
Hamilton, Howard Eugene — 10515 Carnegie av., ALR
Harris, Harold Earl — Cleveland Clinic, ALR
Jackson, Clarence C. R. — Lakeside Hospital, ALR
Jacoby, Mark W. — Hanna bldg., OP
Johnson, Lorand V. — Lakeside Hospital, OP
Kennedy, Roscoe J. — Lt. Comdr. (M.C.), 3277 Bradford road, OP
Keyes, John E. L. — 22199 McCauley road, Shaker Heights, OP
Kinney, Charles Ed. — 10515 Carnegie av., ALR
Large, S. H. — Hanna bldg., ALR
Lemel, Samuel L. — 1030 Euclid av., ALR
McDonald, C. L. — Rose bldg., OALR
Metz, Roy B. — 1422 Euclid av., OP
Metzenbaum, Myron — 10515 Carnegie av., ALR
Mitchell, Horace E. — 14805 Detroit av., ALR
Monson, S. H. — B. F. Keith bldg., OALR
Moore, Paul G. — Republic bldg., OP
Moore, Paul M., Jr. — Cleveland Clinic, ALR
Motto, M. Paul — Rose bldg., OP
Mussun, W. G. — Rose bldg., ALR
Nelson, Charles F. — Schofield bldg., OALR
Phelan, Harold V. — Rose bldg., OP
Pitkin, C. E. — Carnegie Medical bldg., ALR
Quittner, S. S. — 10515 Carnegie av., OALR
Rieger, W. H. — Hanna bldg., ALR
Rosenberger, Harry Clifford — 10515 Carnegie av., ALR
Ruedemann, Albert D. — Cleveland Clinic, OP
Ryall, George W. — 17010 Kinsman road, Shaker Heights, ALR
Shiras, Howard H. — Guardian bldg., OP
Simonds, O. F. — Hanna bldg., OP

OHIO — Continued

Stoffregen, Charles Henry.....10515 Carnegie av., **ALR**
Stotter, Arthur L......1148 Euclid av., **OALR**
Thoburn, Thomas W......1020 Huron road, **ALR**
Tuckerman, W. H......1020 Prospect av., **OALR**
Weidlein, Ivan F......10515 Carnegie av., **ALR**

COLUMBUS

Beatty, H. G......150 E. Broad, **ALR**
Brown, John E......370 E. Town, **OALR**
Clark, I. G......188 E. State, **OALR**
Davies, Morgan C......269 E. State, **OP**
Davis, William C......150 E. Broad, **OP**
Emswiler, Herbert D......370 E. Town, **ALR**
Frost, Albert Dalbey.....150 E. Broad, **OP**
Hauer, A. M......327 E. State, **ALR**
Means, R. G......40 S. Third, **ALR**
Odom, Stanley G......
.....Col. (M.C.), Station Hospital, Fort Hayes, **OALR**
Perry, Claude S......Maj. (M.C.), 40 S. 3rd st., **OP**
Postle, C. D......240 E. State, **ALR**
Prout, A. W......380 E. Town, **OALR**
Sanor, Daniel G., Jr......206 E. State, **ALR**
Sage, Harry M......
.....Lt. Comdr. (M.C.), 2194 N. Parkway Drive, **OP**
Silbernagel, C. E......247 E. State, **OALR**
Stoutenborough, William A......21 E. State, **OALR**
Thomas, Francis W......327 E. State, **OALR**
Timberman, Andrew.....91 Hamilton Park, **OALR**
Troutman, Erwin W......21 E. State, **ALR**

DAYTON

Beavis, James O......900 Fidelity Medical bldg., **ALR**
Binkley, Roy S......Fidelity bldg., **OALR**
Blackburn, W. J......Reibold bldg., **OALR**
Boswell, Robert Echo.....Fidelity bldg., **ALR**
Culler, Arthur M......Fidelity Medical bldg., **OP**
Driscoll, Frederick J......Reibold bldg., **ALR**
Farmer, A. G......Fidelity Medical bldg., **OALR**
Harris, H. B......Fidelity Medical bldg., **OALR**
Kilbourne, P. H......Fidelity Medical bldg., **OALR**
Millette, J. W......117 S. Main, **OALR**
Thomas, Edward Robert.....60 Wyoming, **OP**

ELYRIA

Meredith, Lawrence Cornelius.....124 Middle av., **ALR**

FAIRFIELD

Craddock, Wm. H......Maj. (M.C.), Patterson Field, **ALR**

FINDLAY

Mowry, Ben.....First Nat'l Bank bldg., **ALR**

FREMONT

Riddell, Malcolm M......112½ Front, **ALR**

LA CARNE

Kogut, Lewis V.....Capt. (M.C.), Erie Proving Grounds, **OP**

LAKEWOOD

Ellenberger, Carl.....14805 Detroit av., **OP**
Frackelton, Ralph James.....15701 Detroit av., **ALR**
McGannon, Francis Lambert.....14900 Detroit av., **ALR**
Merica, F. W......14805 Detroit av., **ALR**
Miller, G. Leslie.....14805 Detroit av., **OP**

LIMA

Herr, Albert H......Maj. (M.C.), 212 Steiner bldg., **ALR**
Stueber, F. G......209 W. North, **OALR**
Stueber, Paul J......209 W. North, **OALR**

LORAIN

Monosmith, O. B......O. P. S. bldg., **OALR**

MANSFIELD

Garber, J. M......14 N. Mulberry, **OALR**
Wise, Ralph C......News bldg., **OALR**

MARIETTA

Sauer, W. W......231 Third, **OALR**

MARION

Brickley, Daniel W......Uhler-Phillips bldg., **OALR**
Brickley, Daniel W., Jr......Uhler-Phillips bldg., **ALR**

MASSILLON

Cooper, John Hamilton.....711 Lincoln Way East, **ALR**

MIDDLETOWN

Williams, Walton H......Castell bldg., **OALR**

MONROEVILLE

Bolman, Harold R......6½ S. Main, **OP**

NEWARK

Hatch, C. B......3 W. Church, **OALR**

SALEM

King, Lowell W......470 E. State, **ALR**

SIDNEY

Alvis, Raymond W......Ohio bldg., **OALR**

SANDUSKY

Kramer, Julius C......130 E. Market, **ALR**

SHAKER HEIGHTS

Phillips, Josephine D......16130 Parkland drive, **OP**

SPRINGFIELD

Burgman, James E......8 W. Main, **ALR**
Easton, J. C......First Nat'l Bank bldg., **OALR**
Ernsting, Harry Carl.....8 W. Main st., **OP**
Hartley, F. A......37 S. Kensington, **OALR**
Hogue, D. W......34 W. High, **OALR**
Reuter, Carl H......First Nat'l Bank bldg., **OP**
Yinger, Starling C...Maj. (M.C.), First Nat'l Bank bldg., **ALR**

STEUBENVILLE

Quinn, John L......401 Market, **ALR**
Weinman, Edward B......2513 Sunset blvd., **OP**

TIFFIN

Porter, E. H......85 Madison, **OALR**

TOLEDO

Alderdyce, William W......513 Madison av., **OALR**
Boice, Ralph E......316 Michigan, **OP**
Denyes, Gerald Foster.....316 Michigan, **ALR**
King, Charles R......Spitzer bldg., **OALR**
Lukens, Charles.....316 Michigan, **OALR**
Lukens, John A......316 Michigan, **ALR**
Minns, John Edward.....316 Michigan, **ALR**
Nippe, Leonard.....316 Michigan, **OALR**
Roberts, John L......316 Michigan, **OP**
Skow, John D......2001 Collingwood av., **OP**
Steinfeld, A. L......125 Fifteenth, **OALR**
Winger, Ira B......709 Madison av., **OALR**

VAN WERT

Edwards, Gwilym Austin.....Home Guards bldg., **ALR**
Edwards, Stephen A......Home Guards bldg., **ALR**

WARREN

Stimson, George W......580 Perkins drive, **OALR**

WILLOUGHBY

Waugh, J. M......Ridge road, R.F.D. No. 1, **OALR**

OHIO — Continued

YOUNGSTOWN

Goldcamp, John S.
.....Capt. (M.C.), 810 Dollar Bank bldg., **OP**
Goldcamp, Edward C......Dollar Bank bldg., **ALR**
Hall, Raymond A.....Capt. (M.C.), 3521 Hopkins road, **ALR**
Hartzell, Sol M......260 Tod Lane, **OALR**
Odom, Robert E......24 Wick av., **ALR**
Piercy, Frederick F......Home Savings & Loan bldg., **ALR**
Walker, Orville J......Home Savings & Loan bldg., **ALR**
Wenaas, Elmer J......24 Wick av., **OP**

ZANESVILLE

Culbertson, L. R......Masonic Temple, **OALR**

OKLAHOMA

ADA

Peterson, William G......106 E. 13th, **ALR**

ARDMORE

Henner, Robert.....Capt. (M.C.), 1216 Third st. S.W., **ALR**

CAMP GRUBER

Fowler, Robert H.
.....Maj. (M.C.), C.O. 55th Station Hospital, **ALR**

FORT SILL

McBean, Jas. B......Maj. (M.C.), Station Hospital, **ALR**
McLeod, John.....Maj. (M.C.), Cantonment Hospital, **OP**

FREDERICK

Houser, Ben P......Lt. (M.C.), A.A.F.A.F.S., **ALR**

LAWTON

Schluer, E. P......Maj. (M.C.), 710½ C st., **ALR**

McALESTER

Packard, Louis A......McAlester Clinic, **OALR**

MUSKOGEE

Coachman, Edwin H......Manhattan bldg., **ALR**
Fullenwider, C. M......Barnes bldg., **OALR**

NORMAN

Freimuth, L. E......916 S. Flood, **ALR**

OKLAHOMA CITY

Benham, William L......Medical Arts bldg., **ALR**
Cooper, F. Maxey.....Medical Arts bldg., **OP**
Coston, Tullos O......Medical Arts bldg., **OP**
Hannon, R. Emmet
.....Capt. (M.C.), Oklahoma City Air Depot, **OP**
Macdonald, J. C......301 N. W. 12th, **ALR**
McHenry, L. Chester.....Medical Arts bldg., **ALR**
Patterson, Maj. Gen. Robert U. U.S.A. (Retired)
.....700 N. W. 18th, **ALR**
Randel, Harvey O......Medical Arts bldg., **OP**
Walls, Theodore G......Medical Arts bldg., **ALR**
Watson, O. Alton.....1200 N. Walker, **ALR**

OKMULGEE

Tracewell, George Logan.....Commerce bldg., **ALR**

TULSA

Braswell, James C......Medical Arts bldg., **OALR**
Cook, W. Albert.....Medical Arts bldg., **OALR**
Davis, Arthur Henry.....Medical Arts bldg., **ALR**
Haralson, Charles Herndon.....Medical Arts bldg., **OP**
Huber, Walter A......Medical Arts bldg., **OP**
Mishler, D. L......604 S. Cincinnati av., **ALR**
Roth, Albert W......1616 S. Peoria, **OALR**
Smith, Ruric N......Medical Arts bldg., **ALR**

OREGON

ASTORIA

Spalding, Lansford M......185 Fourteenth, **ALR**

CAMP ADAIR

Kobrak, H. G......Capt. (M.C.), Station Hospital, **ALR**

EUGENE

Norton, Arthur H......1162 Williamette, **OP**

LA GRANDE

Bouvy, L. B......Foley bldg., **OALR**
Ralston, Frank L......Sacajawea Annex, **OALR**

MEDFORD

Meyers, Paul T.
...Capt. (M.C.), 82 General Hospital, Camp White, **ALR**
Young, Clarence M......Drawer 1506, **ALR**

PORTLAND

Bouvy, Harry M......921 S. W. Washington, **OALR**
Boyden, Guy L......1735 N. Wheeler, **ALR**
Browning, Andrew J......Mayer bldg., **OP**
Carruth, Howard Ernest.....Medical Arts bldg., **OALR**
Davis, Ralph F......Medical Arts bldg., **OALR**
Dykman, Augustus Bertram.....833 S. W. 11th, **OP**
Fenton, Ralph A......Medical Arts bldg., **OALR**
Gaston, Ire E......Medical-Dental bldg., **OALR**
Henton, George E......812 S.W. Washington, **OP**
Henton, George H......In Service, 801 Stevens bldg., **OP**
Johnston, Wilson.....Medical Arts bldg., **OALR**
Jordan, Lewis W......Medical Arts bldg., **ALR**
Karkeet, Robert B......833 S.W. 11th av., **ALR**
Kiehle, F. A......1020 S. W. Taylor, **OALR**
Kistner, F. B......Mayer bldg., **ALR**
Lucas, Harold R......Medical-Dental bldg., **OALR**
Lupton, Irving Martin.....Medical Arts bldg., **OALR**
Neely, Paul T......Medical Arts bldg., **ALR**
Petheram, C. C......Medical Arts bldg., **ALR**
Scales, W. M......Maj. (M.C.), Box 569, **OP**
Taylor, E. Merle.....Medical Arts bldg., **OP**
Weeks, John E. (Retired).....360 Edgecliff road, **OP**

SALEM

Clement, L. O......Guardian bldg., **OALR**

WECOMA

Cutler, Franklin E......**ALR**

PENNSYLVANIA

ABINGTON

Hughson, Walter.....Abington Memorial Hospital, **A**

ALIQUIPPA

Thel, Henry C......380 Franklin, **ALR**

ALLENTOWN

Hertz, William J......125 N. 8th, **ALR**
Phillips, Samuel A......127 N. 8th, **OP**
Uhler, S. Mann.....104 N. 8th, **ALR**

ALTOONA

Buzzard, Josiah F......1110 Thirteenth av., **OALR**
Glover, Lewis P......1200 Fourteenth av., **OP**
Hogue, John Dan.....909 Lexington av., **ALR**

ARDMORE

Bostwick, D. S......106 Linwood av., **ALR**
Furlong, Thomas F., Jr......Times Medical bldg., **ALR**

BEAVER

Crumrine, Norman Ross.....595 Third, **OALR**

PENNSYLVANIA — Continued

BEAVER FALLS
Reed, Elmer McCready ..... 1419 7th av., ALR

BETHLEHEM
Chase, W. D. ..... 230 E. Broad, ALR

BRYN MAWR
Harkins, Herbert Perrin, Lt. (M.C.), U.S.N.R., Merion and Montgomery aves., ALR
McAndrews, Leo F. ..... Mermont-Plaza, OP
Snyder, George Gordon ..... Mermont Apts., ALR

BUTLER
Beck, F. W. ..... Capt. (M.C.), Deshon General Hospital, ALR
Goehring, Donald Everett ..... Butler S. & T. bldg., ALR
Hunt, Guy Anderson ..... Butler Co. Nat'l Bank bldg., OP
Schnebly, J. Thomas ..... Capt. (M.C.), Deshon General Hospital, OP

CANONSBURG
Geeseman, George Robert ..... 104 W. Pike st., OP

CHAMBERSBURG
Himelfarb, Hillard Merle ..... Capt. (M.C.), 180 E. Queen, ALR

CHARLEROI
Rowland, Ivo E. ..... 5th and McKean, ALR
Stahlman, Fred C. ..... OALR

CHESTER
Cross, G. H. ..... 525 Welsh, OP

CLEARFIELD
Shaffer, Harry Gard ..... 225 Cherry, ALR

CONNELLSVILLE
Harrison, Fred H. ..... Second Nat'l Bank bldg., OALR
McLellan, Thomas G. ..... First Nat'l Bank bldg., OP

DANVILLE
Davison, Francis Wells ..... Geisinger Hospital, ALR
Jacobs, Clyde Hillock ..... Geisinger Memorial Hospital, OP
Nebinger, Reid ..... 16 W. Market, ALR

DREXEL HILL
Weber, H. C. ..... Comdr. (M.C.), 112 Mason av., OALR

DU BOIS
Beals, C. Wearne ..... 41 N. Brady, ALR
Tyson, John McClure ..... 3 S. Brady, ALR

EASTON
Bloom, C. Hugh ..... 65 N. Third, ALR

ERIE
Delaney, James H. ..... 138 W. 9th, OP
Donovan, John E. ..... 121 W. 9th, ALR
Fust, John H. ..... 231 W. 7th, OALR
Jackson, J. D. ..... 232 W. 8th, OALR
Russell, J. A. Merle ..... 813 Sassafras, OALR
Schlindwein, G. William ..... 138 W. 9th, OALR
Shreve, Owen M. ..... 162 W. 8th, OP

FRANKLIN
Jobson, G. B. ..... West Park and Liberty, OALR
Nordstrom, Chester Albert ..... Exchange Bank bldg., ALR

GREENSBURG
Fairing, John W. ..... 122 N. Main, OALR
McKee, Claud W. ..... Coulter bldg., OALR

HARRISBURG
Dailey, Gilbert L. ..... 618 N. 3rd, OALR
George, Forney Philip ..... 31 S. Front, ALR
Gracey, George F. ..... 209 State, OALR
Zimmerman, J. Landis ..... 207 State, OP

HAZLETON
Reiche, O. C. ..... Markle Bank bldg., OP

HUNTINGDON
Hunt, William T., Jr. ..... 514 Penn, OALR

JENKINTOWN
Cornelius, Chalmers Edgar ..... Lt. Comdr. (M.C.), 119 Summit av., ALR
Kauffman, M. Luther ..... Medical Arts bldg., OP

JOHNSTOWN
Cohen, Jerome Harry ..... Capt. (M.C.), Johnstown Bank & Trust bldg., OP
Griffith, Harold M. ..... Johnstown Bank & Trust bldg., OP
Harris, C. M. ..... Johnstown Bank & Trust bldg., OALR
Hays, Charles E. ..... U. S. Bank bldg., ALR
McCloskey, Paul ..... 338 Locust, ALR
Parker, Ray ..... U. S. Bank bldg., ALR

LANCASTER
Fulton, Harry C. ..... 339 N. Duke, OP
LeFevre, Willis H. ..... 234 N. Duke, ALR
Smith, Ernest Gerard ..... 339 N. Duke, OP

LANSDOWNE
Krall, J. Thomas ..... Maj. (M.C.), 134 Lansdowne court, OP

LATROBE
Mather, Homer Raymond ..... 224 Depot st., ALR

LEWISTOWN
Allis, Paul M. ..... 6 N. Brown, ALR

MAHONEY CITY
Ricchiuti, A. George ..... 23 W. Centre, ALR

MEADVILLE
Hazen, S. Frank ..... 231 Chestnut, ALR

MEDIA
Lewis, Fielding O. ..... (Retired), ALR

MONESSEN
Gemmill, Walter D. ..... 130 Seventh, ALR

MORRISVILLE
Moriconi, Albert F. ..... Maj. (M.C.), 104 E. Ferry road, ALR

NEW KENSINGTON
Steim, J. M. ..... First Nat'l Bank bldg., OP

NORRISTOWN
Beideman, Joseph Ellsworth ..... Montgomery Trust Arcade, OP
LaClair, Chas. H. ..... Maj. (M.C.), 820 W. Marshall, ALR

OIL CITY
Magge, F. Earle ..... 222 Seneca, ALR

PHILADELPHIA
Adler, Francis Heed ..... 313 S. 17th, OP
Annon, W. T. ..... 235 S. 15th, ALR
Appleman, L. F. ..... 308 S. 16th, OP
Babbitt, James A. ..... 1912 Spruce, ALR
Baldwin, Gertrude W. ..... 255 S. 17th st., OP
Balentine, P. L. ..... 1524 Chestnut, OP
Ball, Simon ..... 1920 Pine, ALR
Barlow, Aaron ..... 1307 Spruce, OP
Batson, Oscar V. ..... 133 S. 36th, ALR
Beam, A. Duane ..... Lt. (M.C.), U. S. Naval Hospital, OP
Berner, George Emil ..... 5430 Greene, OP
Bertolet, John Allen ..... 1530 Locust, ALR
Blair, Mortimer W. ..... N.E. cor. 20th and Chestnut, OP
Bricker, Sacks ..... 1101 W. Wyoming av., ALR

PENNSYLVANIA — Continued

Bridgett, Frank Aloysius.....3330 Chestnut st., **ALR**
Briglia, Frank J......819 Wharton, **ALR**
Brown, Clarence J......
.....Capt. (M.C.), U.S.N., Naval Hospital, **ALR**
Brumm, Seth A......Medical Arts bldg., **OALR**
Burns, L. J......1930 Chestnut, **ALR**
Butt, Miriam M......2045 Chestnut, **OP**
Campbell, Edward H......
.....Lt. Comdr. (M.C.), U.S.N.R., In Service, **ALR**
Campbell, Sherburne.....
.....Lt. Comdr. (M.C.), U. S. Naval Hospital, **OP**
Carpenter, Chapin.....1930 Chestnut, **OP**
Clerf, Louis Henry.....1530 Locust, **ALR**
Coates, George M......1721 Pine, **ALR**
Cohen, Herman B......255 S. 17th, **ALR**
Cohen, Louis B......1320 Arrott, **OP**
Cohen, Samuel.....1920 Pine, **ALR**
Cowan, Alfred.....1930 Chestnut, **OP**
Creighton, William J......37 S. 20th, **OP**
Davis, T. Carroll.....3128 N. Broad, **ALR**
Davis, Warren B......135 S. 18th, **ALR**
Deichler, L. Waller.....1930 Chestnut, **OALR**
Dickson, Robert L......4803 Penn, **ALR**
Dintenfass, Henry.....1305 Spruce, **ALR**
Donnelly, Edward J......1321 Spruce, **OP**
Dublin, George Joseph.....1930 Chestnut, **OP**
Elfman, Louis Kenneth.....6330 Lebanon av., **ALR**
Eraner, Matthew S......1915 Spruce, **ALR**
Fewell, A. G......1924 Pine, **OP**
Fox, C. Calvin.....1806 Spruce, **ALR**
Fry, W. E......1819 Chestnut, **OP**
Gibson, Glen G......255 S. 17th, **OP**
Goddard, H. M......1531 Spruce, **ALR**
Gordon, William.....1721 Pine, **ALR**
Gouterman, Joseph I......
..Maj. (M.C.), 344 W. School Lane (Germantown), **OP**
Gowens, Henry Lytle, Jr......123 S. 23rd st., **OP**
Griscom, J. Milton.....255 S. 17th, **OP**
Hawman, Erle G......4051 N. Broad, **OP**
Hebble, Howard M......
.....Lt. (j. g.) (M.C.), U. S. Naval Hospital, **ALR**
Head, C. R......1205 Spruce, **OP**
Hewson, William.....6013 Greene (Germantown), **ALR**
Hirst, Otto C......255 S. 17th, **ALR**
Hitschler, William Jacob.....8540 Germantown av., **ALR**
Houser, Karl M......2010 Spruce, **ALR**
Howland, Alvin Wilcox.....Maj. (M.C.), 255 S. 17th, **OP**
Hunter, R. J......
.....Lt. Comdr. (M.C.), U.S.N.R., 928 N. 63rd, **ALR**
Husik, D. N......1820 Spruce, **ALR**
Ivy, Robert H......1930 Chestnut, **ALR**
Jackson, Chevalier.....255 S. 17th, **L**
Jackson, Chevalier L......255 S. 17th, **L**
Johnson, George Ernest.....5341 Chester av., **ALR**
Kasper, Kelvin A......135 S. 18th, **ALR**
Katz, Harry Kolman.....1206 Cottman st., **OP**
Kaufman, A. Spencer.....1923 Spruce, **ALR**
Kelly, George F. J......37 S. 20th, **OP**
Krauss, Frederick.....1930 Chestnut, **OALR**
Krewson, William Egbert, 3rd.....1930 Chestnut, **OP**
Lawrence, Granville A......Medical Arts bldg., **OP**
Lederer, Francis L......
.....Lt. Comdr. (M.C.), U. S. Naval Hospital, **ALR**
Lehrfeld, Louis.....1321 Spruce, **OP**
Lillie, W. Ivan.....255 S. 17th, **OP**
Loftus, John Edward.....1930 Chestnut, **ALR**
Lukens, Robert McD......1923 Spruce, **ALR**
Luongo, R. A......2054 Locust, **ALR**
McCarthy, C. T......1810 Pine, **ALR**
McDonald, P. Robb.....Capt (M. .C.). 255 S, 17th, **OP**
Mackenzie, George W......269 S. 19th, **OALR**
Mann, Benjamin H......1930 Chestnut, **OP**
Miller, M. Valentine.....114 W. Phil-Ellena (Mt. Airy), **ALR**
Mitchell, E. K......710 W. Lehigh av., **ALR**
Mullen, Carroll R......2025 Locust, **OP**
Olsho, S. L......235 S. 15th, **OALR**
Persky, Abram H......1723 Pine, **ALR**
Pryor, Charles Allen.....1910 Spruce, **ALR**
Reese, Warren S......1901 Walnut, **OP**
Richardson, George A......1532 Overington, **ALR**
Ridpath, Robert F......1737 Chestnut, **LR**
Rommel, John C......4601 N. Broad, **OP**
Rosenthal, Joseph M......1737 Chestnut, **OP**
Rudolphy, Jay Besson.....230 S. 21st, **OP**
Sartain, Paul J......2006 Walnut, **OP**
Scarlett, Hunter W......230 S. 21st, **OP**
Seltzer, Albert Pincus.....1332 N. Franklin st., **ALR**
Shannon, C. E. G......1930 Chestnut, **OP**
Shipman, James S......255 S. 17th, **OP**
Shuster, Benjamin Harrison.....1824 Pine, **ALR**
Simkins, James J......1737 Chestnut, **ALR**
Skillern, S. R., Jr......1734 Pine, **ALR**
Sloane, Henry O......1717 Pine, **OP**
Spaeth, Edmund B......1930 Chestnut, **OP**
Spencer, W. H......1830 S. Rittenhouse sq., **ALR**
Stiles, Charles M......1831 Chestnut, **OP**
Stockwell, Eunice LeBaron.....2018 Pine st., **OP**
Sutherland, William S......1930 Chestnut, **OP**
Swalm, Charles Joseph.....1330 W. Rockland, **ALR**
Tasman, Isaac S......Medical Arts bldg., **OP**
Towson, Charles E......Germantown Prof. bldg., **ALR**
Tucker, Gabriel.....250 S. 18th, **LB**
Wagers, Arthur J......1429 Spruce, **ALR**
Waldman, Joseph.....
..Maj. (M.C.), Park Manor Apts., 4000 Girard av., **OP**
Walsh, Thomas J......2059 Parrish, **ALR**
Weinstein, M. A......1530 Locust, **ALR**
Whelan, G. L......2100 Walnut, **ALR**
Whelan, William F......2004 Walnut, **ALR**
Williams, Horace J......5908 Greene (Germantown), **ALR**
Winston, Julius.....2021 Spruce, **ALR**
Zacks, Myron Albert.....1737 Chestnut, **ALR**
Zentmayer, William.....1930 Chestnut, **OP**

PHOENIXVILLE

Abbott, A. J......
.....Capt. (M.C.), Valley Forge General Hospital, **OP**

PITTSBURGH

Barnatz, C. F......Park bldg., **OP**
Billings, Robert J......500 Penn av., **OP**
Blair, W. W......Woodland road, (6), **OP**
Boaz, Emmett D......3710 Fifth av., **ALR**
Carson, W. E......Jenkins Arcade, **OP**
Conway, John M......Jenkins Arcade, **ALR**
Cooper, Roy C......510 S. Aiken av., **ALR**
Curry, Glendon E......Diamond Bank bldg., **OP**
Day, Kenneth M......121 University pl., **ALR**
De Stio, Daniel Stephen.....Highland bldg., **ALR**
Dunbar, John Charles.....Empire bldg., **OP**
Fischer, N. Arthur.....Union Trust bldg., **ALR**
Friedman, Louis Leonard.....6078 Jenkins Arcade, **ALR**
Gross, Julius E......3401 Fifth av., **ALR**
Hagemann, John A......Highland bldg., **ALR**
Hamilton, Richard G......Medical Arts bldg., **ALR**

PENNSYLVANIA — Continued

Hampsey, Joseph William....806 May bldg., ALR
Hinderer, Kenneth H....Medical Arts bldg., ALR
Hogsett, Smith F., Jr.
....Lt. (j. g.) (M.C.), U.S.N.R., 7144 Meade st., OP
Jennings, Charles W....121 University pl., OP
Krebs, Adolph....500 Penn av., OP
Larimore, Fred Campbell....500 Penn av., OP
Linn, J. G....Jenkins Arcade, OP
McCaslin, Murray F....Union Trust bldg., OP
McCollough, T. B....121 University pl., ALR
McCready, J. Homer....121 University pl., ALR
MacLachtan, A. A....May bldg., ALR
Markel, J. Clyde....806 Pitt Bank bldg., OP
Marshall, Watson....121 University pl., ALR
Moon, Seymour B....429 Pennsylvania av., OP
Morgan, Irving Joseph....Jenkins Arcade bldg., OP
Pachtman, Isadore....Jenkins Arcade bldg., OP
Patterson, Ellen J....(Retired), 504 Neville, ALR
Perrone, Joseph Addison....Mercy Hospital, ALR
Plumer, John S....121 University pl., OP
Ratner, Simon Harry....3401 Fifth av., ALR
Reed, Charles L....Jenkins Arcade, OP
Rhodes, David Henry....Clark bldg., OP
Rimer, Frank H....Jenkins Arcade, ALR
Rohm, Robert Franklin....7026 Jenkins Arcade, OP
Shaffer, D. H....7036 Jenkins Arcade, OP
Shepard, Warren Brundage....Jenkins Arcade, ALR
Shuman, G. H....351 Fifth av., OP
Simpson, John Reid....Medical Arts bldg., ALR
Statti, Louis William....Jenkins Arcade, OP
Stieren, Edward....Union Trust bldg., OP
Taylor, M. C....May bldg., OP
Thorpe, H. E....Jenkins Arcade, OP
Todd, Grover Cleveland....Jenkins Arcade, ALR
Turner, H. H....Clark bldg., OP
Weisser, E. A....May bldg., OP
Yost, Walter Milton....Westinghouse bldg., OP

PITTSTON

Gagion, Thomas Reed....23 Broad, OP
Gallizzi, Vincent D....51 S. Main st., ALR

POTTSTOWN

Hancox, Cecil William....Bahr Arcade bldg., ALR

POTTSVILLE

Cress, William J....318 W. Market, ALR
Mengel, Sterling Frederic
....Lt. Comdr. (M.C.), 612 W. Market, OP

READING

Bankes, Claude W....212 N. 6th, OP
Craig, Paul C....232 N. 5th, OP
High, Isaac B....326 N. 5th, ALR
Landis, James E....232 N. 6th, ALR
Penta, Michael J....312 N. 5th, ALR
Rhode, Homer J....220 N. 6th, OP
Rhode, Solon L....238 N. 6th, OALR
Wotring, John M....220 N. 6th, OP

ROCHESTER

Miksch, Henry Fred....209 S. Park, ALR

SCRANTON

Bishop, Frederick J....Medical Arts bldg., ALR
Corser, J. B....415 Mulberry, OP
Curtin, Eugene A....506 Spruce, OALR
Jordan, James Shannon....Medical Arts bldg., OP
Williams, M. M....Medical Arts bldg., OP

SHARON HILL

Tomlinson, John Wesley
....Lt. Comdr. (M.C.), 900 Chester Pike, OP

SHENANDOAH

Monahan, James J....31 S. Jordin, ALR

SUMMIT HILL

Bonner, William Richard....3 W. White st., OP

TURTLE CREEK

Hunter, James F....503 Penn av., ALR

UPPER DARBY

Kennedy, Patrick James....6910 Market, OP
Moore, George H....101 Long Lane, OALR
Rankin, Charles A....Ludlow and Heather road, OP

WARREN

Anderson, Edwin R....220 Liberty, OALR
Ball, Michael V....316 Hazel, OALR

WASHINGTON

McCullough, Clarence Joseph....Washington Trust bldg., ALR
McKennan, James W....Washington Trust bldg., OP
McMurray, J. B....Washington Trust bldg., OALR

WILKES-BARRE

Buckman, Lewis T....53 S. Franklin, OALR
Buckman, Samuel Thompson....70 S. Franklin, ALR
Connole, Joseph V....Miners Nat'l Bank bldg., OP
Kistler, O. F....43 N. Franklin, OP AR
Marsden, W. C....99 N. Franklin, OALR
Schappert, N. Louis....59 S. Washington, OALR

WILLIAMSPORT

Decker, Philip Harold....25 W. 3rd st., ALR

YORK,

Fisher, Lawrence Christian....459 W. Market, OP
Grove, Bruce Alexander....141 E. Market st., OP
Klinedinst, John Fred....220 S. George, OPA

RHODE ISLAND

DAVISVILLE

Wall, John W....Lt. (M.C.), N.C.T.C. Dispensary, ALR

FORT GETTY

Millett, Frank A....Lt. Col. (M.C.), Station Hospital, OALR

NEWPORT

Jacoby, Douglas P. A....106 Touro, OAL
Meek, R. E....Comdr. (M.C.), U. S. Naval Hospital, OP
Moyle, Eugene H.
....Lt. Comdr. (M.C.), U. S. Naval Hospital, ALR

PROVIDENCE

Adams, Frank M....122 Waterman, ALR
Bolotow, Nathan A....126 Waterman, ALR
Dimmitt, Frank W....78 Waterman, OALR
Grossman, Herman P....210 Angell, OP
Harvey, N. Darrell....112 Waterman, OALR
McCurdy, Gordon Judson....198 Angell, ALR
MacCardell, Frank Cameron....193 Waterman, ALR
Messinger, H. C....210 Angell, OP
Muncy, William M....162 Angell, OALR
Porter, L. B....199 Thayer, OALR
Winkler, Herman A....224 Thayer, ALR

QUONSET POINT

Hurd, Allan C., Lt. (M.C.), U.S.N.R.,
c/o Dispensary, Naval Air Station, ALR

SOUTH CAROLINA

ANDERSON

Gaines, Thomas Rucker.....Professional bldg., OP

BEAUFORT

Macnie, John P.....P. O. Box 175, OP

CAMP CROFT

Fairfax, Kenneth I.....Maj. (M.C.), Station Hospital, OP
Holmes, W. W.....Lt. Col. (M.C.), Station Hospital, ALR

CHARLESTON

Evatt, Clay W.....112 Rutledge av., ALR
Hanckel, Richard White, Jr.....96-A Bull st., ALR
Hope, Robert Meek.....108 Rutledge av., ALR
Pritikin, Roland I.....
.....Lt. Col. (M.C.), Stark General Hospital, OP
Schoolman, Joseph G.....
.....Capt. (M.C.), Stark General Hospital, ALR
Smith, Josiah Edward.....60 Meeting, OALR
Swift, Donald E.....Stark General Hospital, OP
Townsend, John Ferrars.....85 Wentworth, OALR
Trueman, Robert Heiserman.....
.....Capt. (M.C.), Stark General Hospital, OP

COLUMBIA

Bristow, Walter J.....1517 Hampton, OALR

FORT JACKSON

Smith, Jay D.....
.....Maj. (M.C.), Box 6, Station Hospital No. 2, ALR

GREENVILLE

Krimsky, Emanuel.....
.....Maj. (M.C.), Base Hospital, Army Air Base, OP
Sanders, John Levis.....222 N. Main, ALR

PARIS ISLAND

Hutchison, Lloyd S.....
.....Lt. Comdr. (M.C.), U. S. Naval Hospital, ALR
Livshin, Norman...Lt. Comdr. (M.C.), Naval Hospital, ALR

ROCK HILL

Macdonald, Roderick.....330 E. Main, ALR

SUMTER

Walker, Robert Murdoch.....
.....Lt. (M.C.), U.S.N.R., 10 E. Calhoun st., ALR

SOUTH DAKOTA

ABERDEEN

Bunker, Paul Gordon.....Newberry bldg., ALR
Rudolph, Edward.....Newberry bldg., OP

MITCHELL

Mabee, Oscar Judson.....Medical Arts bldg., ALR

PIERRE

Robbins, Clarence Edward.....111 S. Huron av., ALR

SIOUX FALLS

Butler, Harry.....Maj. (M.C.), A.F.T.T.S., ALR
Gregg, John B.....Security Nat'l Bank bldg., OALR
Keller, S. A.....303 S. Minnesota av., OALR

YANKTON

Hohf, Julius A.....Clinic bldg., OALR

TENNESSEE

CHATTANOOGA

Hogshead, J. M.....602 Georgia av., OALR
Isbell, D.....552 McCallie av., ALR
Lassiter, Chester L.....Hamilton Nat'l Bank bldg., ALR
Lawwill, Stewart.....Medical Arts bldg., OALR
Long, Samuel Herman.....Medical Arts bldg., ALR
Steele, Willard.....721 Broad, OALR

CLARKSVILLE

Wilson, Paul Edwin.....408 Franklin, OP

CLEVELAND

Ferguson, E. R.....Physicians and Surgeons Hospital, ALR

CROSSVILLE

Mercer, Ray.....Lt. Col. (M.C.), P.W.I.C. Hospital, OALR

DYERSBURG

Brewer, John D.....Braid, Brewer General Hospital, OALR

JOHNSON CITY

Jones, U. G.....Cor. Market and Boone, ALR
McKee, Thomas Preston.....Jones Hospital, ALR

KINGSPORT

Hodge, James Vernon.....105 Market, ALR

KNOXVILLE

Cunningham, Henry Kelso.....Medical Arts bldg., ALR
Kincaid, J. H.....421 W. Church av., OALR
Leach, Robert Swepson.....422 W. Cumberland av., OP
Le Tellier Forrest S.....Doctors bldg., OP
Miller, Edwin Ephron.....Medical Arts bldg., ALR
Potter, William W.....501 Walnut, OALR

MARYVILLE

Burchfield, George W.....309 Court, ALR

MEMPHIS

Anderson, R. G.....
.....Capt. (M.C.), Kennedy General Hospital, OP
Anthony, Daniel Harbert.....Exchange bldg., OP
Blassingame, C. D.....899 Madison av., ALR
Blue, J. B.....62 N. Main, OALR
Ellett, E. C.....Exchange bldg., OP
Evans, S. S.....Physicians & Surgeons bldg., ALR
Farrington, P. M.....89 Porter bldg., ALR

Harris, J. Harley.....Methodist Hospital Doctors bldg., ALR
Lasky, Mortimer A., Maj. (M.C.),
Base Hospital, A.T.C., Municipal Airport, OP

Levy, Louis.....Commerce Title bldg., ALR
Lewis, A. C.....130 Madison av., OALR
Lewis, Philip Meriwether.....Exchange bldg., OP
McKinney, James Wesley.....Exchange bldg., OP
Minor, H. F.....Commerce Title bldg., OALR
Richardson, G. A.....U. S. Naval Hospital, ALR
Rychener, Ralph O.....130 Madison av., OP
Sanders, Samuel H.....1089 Madison av., ALR
Seligstein, Milton B.....Union Planters Bank bldg., OALR
Shea, J. J.....1018 Madison av., ALR
Simpson, W. Likely.....Physicians & Surgeons bldg., OALR
Stanford, J. B.....899 Madison av., OP
Stinson, William David.....899 Madison av., ALR

NASHVILLE

Bryan, James Leslie.....Bennie-Dillon bldg., OALR
Cullom, M. M.....Bennie-Dillon bldg., OALR
Hasty, F. E.....3631 West End av., ALR
Hollabaugh, Charles F.....706 Church, OP
Kennon, W. G.....Doctors bldg., OALR
Manees, Guy Milford.....119 7th av. N., ALR
Orr, Eugene.....Doctors bldg., ALR
Smith, Henry C.....c/o Alex Stevenson, Am. Nat'l Bank, OP
Sullivan, Robert E.....Doctors bldg., OP
Warner, Robert Jay.....706 Church, OP
Wilkerson, William W., Jr.....Bennie-Dillon bldg., ALR

TENNESSEE — Continued

SMYRNA

Aronson, S. S......Capt. (M.C.), Station Hospital, **ALR**

TULLAHOMA

Merkel, Byron M., Maj. (M.C.),
75th Observation Group, Army Air Base, **ALR**

TEXAS

AMARILLO

Crume, John James.....724 Polk, **ALR**
Duncan, Robert Austin.....Amarillo bldg., **ALR**
Streit, August J......724 Polk, **ALR**

AUSTIN

Hilgartner, H. L., Jr...Maj. (M.C.), 2808 Bowman road, **OP**
Key, Samuel N......Norwood bldg., **OALR**
Robison, J. T......Norwood bldg., **ALR**
Suehs, Oliver W......Norwood bldg., **ALR**

BIG SPRING

Malone, P. W......811 Main, **ALR**

BROWNWOOD

Anderson, W. B......309 W. Anderson, **OALR**

CAMP BARKELEY

Anneberg, A. Rees.Capt. (M.C.), 37th Station Hospital, **ALR**
Baird, Charles G...Col. (M.C.), 130th Station Hospital, **OALR**
Berns, Charles..Maj. (M.C.), 34th Evacuation Hospital, **ALR**
Deutsch, William, Jr....Capt. (M.C.), Station Hospital, **ALR**

CAMP BOWIE

Balding, L. Grant.....17th Field Hospital, **OP**
Mosher, Henry A......Capt. (M.C.), Station Hospital, **OP**
Siewart, Otto L..Capt. (M.C.), General Hospital No. 32, **OP**

CAMP HOOD

Faier, Samuel Z......Lt. (M.C.), Station Hospital, **OALR**

CAMP HOWZE

Heare, Louis C......Maj. (M.C.), Station Hospital, **OP**

CORPUS CHRISTI

Riggs, R. H...Lt. Comdr. (M.C.), U. S. Naval Hospital, **ALR**
Smith, Alma C......Capt. (M.C.), Naval Air Station, **OALR**
Stroud, Sanders Key.....Medical Professional bldg., **OP**

DALLAS

Brannin, Daniel.....Medical Arts bldg., **OP**
Cary, E. H......Medical Arts bldg., **OALR**
Cox, Kelly.....Medical Arts bldg., **OP**
Darrough, L. E......4105 Live Oak, **ALR**
Harrington, Silas Frederick.....Medical Arts bldg., **ALR**
Knowles, W. Mood.....Medical Arts bldg., **ALR**
McCullough, Malcolm Kelley.....Medical Arts bldg., **OP**
McLaurin, J. G......4710 Munger av., **OALR**
Marchman, Oscar M......5328 Live Oak, **ALR**
Nelson, Leo A......4105 Live Oak, **ALR**
Newton, F. H......Medical Arts bldg., **OALR**
Quinn, Lester Hale.....4105 Live Oak, **OP**
Sellers, L. M......1719 Pacific av., **ALR**
Singleton, John Dudley.....1719 Pacific av., **ALR**
Wilkinson, Wallace Bond.....521 E. 10th, **ALR**

DEL RIO

Mertins, Paul S., Jr......Capt. (M.C.), A.A.F.A.F.S., **ALR**

DUNCAN FIELD

Moran, Charles T......Capt. (M.C.), Station Hospital, **OP**

EL PASO

Filmer, George A......
...Capt. (M.C.), Wm. Beaumont General Hospital, **OP**
Fitz-Hugh, G. S......
...Capt. (M.C.), Wm. Beaumont General Hospital, **ALR**
Rubright, LeRoy.....
...Capt. (M.C.), Wm. Beaumont General Hospital, **ALR**
Schuster, Frank P......First Nat'l Bank bldg., **OALR**
Schuster, S. A......First Nat'l Bank bldg., **ALR**
Vandevere, William E......First Nat'l Bank bldg., **OALR**

FORT WORTH

Eschenbrenner, John William.....
.....Maj. (M.C.), 3600 Country Club Circle, **ALR**
Goodman, Thomas L......1601 Clover Lane, **ALR**
Gough, Roy H......600 W. 10th, **OALR**
Jackson, Atras E......Medical Arts bldg., **OP**
Richardson, James Joseph.Cook Hosp. and Beall Clinic, **ALR**
Schenck, C. P......Medical Arts bldg., **OALR**
Spearman, M. P. S......U. S. Marine Corps Air Base, **ALR**

GAINESVILLE

Mead, Ernest Charles.....6½ E. California, **ALR**

GALVESTON

McReynolds, Geo. S., Jr......927 Strand, **ALR**
Potter, William Bently.....
.....University of Texas Medical College, **OP**
Quinn, Robert E......4014 Quenuet, **OP**
Sykes, Clarence Sherbourne.....2201 Market, **OP**

GREENVILLE

Gill, E. K......Maj. (M.C.), Mators Army Air Field, **ALR**
Votaw, Robert E......Maj. (M.C.), Station Hospital, **ALR**
Ward, James W......2607 Lee, **ALR**

HONDO

Hitz, John B......Maj. (M.C.), A.A.F.N.S., **OP**

HOUSTON

Alexander, Charles S..Maj. (M.C.), Medical Arts bldg., **OP**
Bressler, J. L......Medical Arts bldg., **OP**
Cody, Claude C......1304 Walker av., **ALR**
Daily, Louis.....Medical Arts bldg., **OALR**
Daily, Ray K......Medical Arts bldg., **OALR**
Dickson, J. Charles.....Medical Arts bldg., **ALR**
Elles, Norma B......Niels Esperson bldg., **OALR**
Foster, J. H......1304 Walker av., **ALR**
Goar, Everett Logan.....1300 Walker av., **OP**
Griffey, Edward W......Medical Arts bldg., **OALR**
Haden, Henry C......1914 Travis, **OP**
Harris, Herbert Houston.....
.....Capt. (M.C.), Mellie Esperson bldg., **ALR**
Janse, Hector Mason.....3717 Main, **ALR**
Logue, Lyle J......1304 Walker av., **ALR**
Ralston, Wm. W......1300 Walker av., **OP**
Schultz, Jacob F......Capt. (M.C.), 1304 Walker av., **OP**
Spencer, Walter C. E......Medical Arts bldg., **OP**
Stough, John Trew.....Medical Arts bldg., **OP**
Vanzant, Thomas J......Medical Arts bldg., **OP**

LONGVIEW

Hurst, V. Reeves.....Hurst Clinic, **OALR**
Jaffe, Benjamin.....
.....Capt. (M.C.), Harmon General Hospital, **OP**
Roberts, J. D., Jr......Lt. (M.C.), U.S.N.R., **ALR**

LUBBOCK

Hutchinson, Benjamin B....Lt. (M.C.), 1301 Broadway, **OP**
Malone, F. B......2609 19th, **ALR**

McKINNEY

Lowenthal, Gerson.....
.....Capt. (M.C.), Ashburn General Hospital, **ALR**

MARFA

Barr, William T......Capt. (M.C.), Air Base Hospital, **ALR**

TEXAS — Continued

PAMPA
High, Clifton E......Combs Worley bldg., **OP**

PARIS
Hunt, Thomas Ewell.....First Nat'l Bank bldg., **ALR**

PORT ARTHUR
Vaughan, Benjamin H......Adams bldg., **ALR**

RANDOLPH FIELD
Campbell, Paul A......
.....Lt. Col. (M.C.), School of Aviation Medicine, **ALR**
Simpson, Robert K......Station Hospital, **OP**

SAN ANGELO
Windham, Robert Edward....234 W. Beauregard blvd., **OALR**

SAN ANTONIO
Atkinson, D. T......Medical Arts bldg., **OALR**
Boehs, Charles John.....Medical Arts bldg., **ALR**
Champion, Albert N......Medical Arts bldg., **ALR**
Cone, Alfred J......
Maj. (M.C.), San Antonio Aviation Cadet Center, **ALR**
Dumas, Edward DeVan.....Medical Arts bldg., **ALR**
Gill, William Davis.....705 E. Houston, **OALR**
Muldoon, Wilfred E......Nix Professional bldg., **OP**
Owsley, Robert W......Capt. (M.C.), 602 Ogden Lane, **ALR**
Parrish, Robert E......Medical Arts bldg., **OALR**
Phillips, James Winston.....
.....Lt. (M.C.), Brooks General Hospital, **ALR**
Rosebrough, Frank Henry.....South Texas Bank bldg., **OP**
Russell, Daniel A......Medical Arts bldg., **ALR**
Sykes, Edwin Meredith.....Nix Professional bldg., **ALR**
Townes, C. D......
.....Maj. (M.C.), Station Hospital, S.A.A.C.C., **OP**
Walker, C. E., Jr......Maj. (M.C.), S.A.A.C.C., **OP**

SHEPPARD FIELD
Montgomery, John L......Station Hospital, **OP**
Page, Robert O......Maj. (M.C.), Station Hospital, **ALR**

SHERMAN
Thomas, W. Maxwell.....
.....Lt. Comdr. (M.C.), 437 W. Houston, **OP**
Waltz, Harold D......Maj. (M.C.), Perrin Field, **ALR**

STEPHENVILLE
Terrell, Vance.....Stephenville Hospital, **ALR**

TAYLOR
Alexander, Margaret Henrietta.....720 Talbot, **ALR**

TEMPLE
Dowling, J. D......
.....Capt. (M.C.), McCloskey General Hospital, **ALR**
McDavitt, Bertha Schenck Stecker.....Main and av. A, **ALR**
Norwood, Eugene P......c/o Kings Daughters Clinic, **ALR**
Watkins, V. O......
.....Capt. (M.C.), McCloskey General Hospital, **OP**
Woodson, B. Palmer.....Woodson Hospital, **OALR**
Woodson, Warren Burbank.....Woodson Hospital, **OALR**

TEXARKANA
Fuller, T. E......209 Pine, **OALR**
Mann, Albert H......2324 Glendale av., **ALR**
Roberts, A. Warren.....209 Pine, **OALR**

TYLER
Vaughn, E. H......Peoples Nat'l Bank bldg., **OALR**

WACO
Aynesworth, H. T......Medical Arts bldg., **OALR**
Burgess, John Louis.....Medical Arts bldg., **ALR**
Pryor, Will R......824 N. 18th, **OP**
Spencer, Shelby Cruthirds.....Professional bldg., **OALR**
Woolsey, William Jinkins.....212 N. 7th st., **OP**

WAXAHACHIE
Donnell, Herbert.....507 W. Main, **ALR**

WICHITA FALLS
Nail, James B......1300 Eighth, **ALR**
Steffensen, W. H......
....Capt. (M.C.), Station Hospital, Sheppard Field, **ALR**

UTAH

FORT DOUGLAS
Swigert, John L......Maj. (M.C.), Station Hospital, **OP**

OGDEN
Harlowe, H. D......
.....Capt. (M.C.), Station Hospital, Hill Field, **ALR**
Hetzel, C .C......Eccles bldg., **OALR**

PROVO
Oaks, Lewis Weston.....33 E. 2nd S., **OALR**

SALT LAKE CITY
Le Compte, E. D......9 Exchange place, **OALR**
Maw, Raymond B......699 East South Temple, **ALR**
Neher, Edwin Manson.....Boston bldg., **OP**
Palmer, Bascom W......Boston bldg., **OP**
Smith, William Leroy.....Medical Arts bldg., **OP**
Stauffer, F. Leaver.....Medical Arts bldg., **ALR**
Welsh, Thomas F......First Nat'l Bank bldg., **OALR**
White, Vivian P......Tribune Telegram bldg., **ALR**

VERMONT

BARRE
Jarvis, De Forest G......Quarry Bank bldg., **ALR**

BURLINGTON
Arnold, Francis J......182 Pearl, **OALR**
Twitchell, Emmus George.....266 Main, **OALR**

MONTPELIER
Bertrand, George Joseph.....73 Main st., **OP**

RUTLAND
Marshall, George Guerin.....Gryphon bldg., **OALR**

ST. JOHNSBURY
Cramton, Edward Allen.....33 Main, **OALR**

VIRGINIA

BLACKSTONE
Kaplan, Samuel...Maj. (M.C.), Camp Pickett Hospital, **ALR**

CHARLOTTESVILLE
Burton, Edwin Weisman.....Y. M. C. A. bldg., **OP**
Krimsky, Joseph.....Rio road, **OALR**
Woodward, Fletcher Drummond.....104 E. Market, **ALR**

COVINGTON
Winn, Thomas Meredith.....103 Maple av., **OALR**

DANVILLE
Dickerson, William E......Masonic Temple, **ALR**
McGovern, Francis H......Masonic Temple bldg., **ALR**

FORT BELVOIR
Hayes, Merrill B......Capt. (M.C.), Station Hospital, **ALR**

FREDERICKSBURG
Holloway, J. Minor.....1200 Prince Edward, **ALR**

HARRISONBURG
Preston, H. Grant.....Professional bldg., **ALR**

VIRGINIA — Continued

LYNCHBURG

Gorman, James R.....Allied Arts bldg., **ALR**
Holland, John Gill....725 Church, **ALR**
Morrison, Frederick M.....Allied Arts bldg., **OP**

NEWPORT NEWS

Hankins, George Geddy....Medical Arts bldg., **ALR**
Poindexter, Frank Wilmore....Medical Arts bldg., **ALR**

NORFOLK

Benson, C. E.....Lt. Comdr. (M.C.), 1021 Redgate av., **ALR**
Cooley, Carl Conrad....Medical Arts bldg., **ALR**
Knapp, Arthur A.....
....Comdr. (M.C.), N.O.B., U. S. Naval Hospital, **OP**
Leamer, Bruce V.....
....Patrol Wing No. 8, Naval Air Station, **ALR**
O'Rourke, Carroll....
Lt. Comdr. (M.C.), U. S. Naval Hospital N.O.B., **ALR**
Provost, Pierre E.....Lt. (M.C.), Naval Air Station, **ALR**
White, Joshua Warren....Medical Arts bldg., **OALR**

PORTSMOUTH

Cowen, Jack P.....Lt. (M.C.), U. S. Naval Hospital, **OP**
Fuhring, S. A.....Norfolk Naval Hospital, **ALR**
O'Kane, Geo. H.....U. S. Naval Hospital, **ALR**
Hooker, James F.....
....Capt. (M.C.), U.S.N.R., Norfolk Navy Yard, **ALR**
Young, Charles A., Comdr. (M.C.),
Norfolk Naval Hospital, **OP**

PULASKI

Smith, Richard O.....73 Third av. N.W., **ALR**

QUANTICO

Albright, P. M.....
....Capt. (M.C.), U.S.N., U. S. Naval Hospital, **OALR**
Benkwith, Karl B., Lt. (M.C.),
U. S. Naval Hospital, Marine Barracks, **OP**
Stewart, James K., Lt. Comdr. (M.C.),
Marine Barracks, U. S. Naval Hospital, **ALR**

RICHMOND

Courtney, Robert H.....Professional bldg., **OP**
Gatewood, E. T.....Professional bldg., **ALR**
Hughes, T. E.....1000 W. Grace, **ALR**
Pastore, Peter Nicholas....
....Medical College of Virginia Hospital, **ALR**
Wallerstein, Emanuel U.....Professional bldg., **ALR**

ROANOKE

Gill, E. G.....711 S. Jefferson, **ALR**
Hamlin, Fred Eugene....Medical Arts bldg., **ALR**
Hatcher, Wm. F.....Medical Arts bldg., **OP**
Stone, Harry B.....Medical Arts bldg., **ALR**

STAUNTON

Fisher, Guy Rothwell....1 E. Beverley st., **ALR**

WILLIAMSBURG

Abbott, E. A.....Lt., Camp Peary, **ALR**

WINCHESTER

Hawthorne, Allen T.....110 N. Braddock, **ALR**
McGuire, H. H.....105 N. Braddock, **OP**

WASHINGTON

BELLINGHAM

Howe, Spencer S.....Bellingham Nat'l Bank bldg., **OALR**

BREMERTON

Johnson, David M...Lt. Comdr. (M.C.), Navy Hospital, **ALR**
Jones, Lester T...Comdr. (M.C.), Naval Base Hospital, **ALR**
Peterson, John C., Lt. Comdr. (M.C.),
Yard Dispensary, Navy Yard, Puget Sound, **OP**

COLFAX

Henry, Robert....Capt. (M.C.), 322 E. James st., **OP**

ELLENSBURG

Hicks, William Wynne....202 E. 6th st., **ALR**

EVERETT

Holstein, Theodore....Capt. (M.C.), Paine Field, **OP**
Murphy, Albert B.....Medical and Dental bldg., **ALR**
Powers, H. W.....
....Maj. (M.C.), Station Hospital, Paine Field, **ALR**
Secoy, Harry R.....Central bldg., **ALR**

FORT GEORGE WRIGHT

Baum, W. W...Maj. (M.C.), Army Air Base Hospital, **OALR**

LONGVIEW

Barton, John F.....1346 Fourteenth av., **ALR**

PASCO

Flynn, J. Donald....
....Lt. (M.C.), Naval Air Station Dispensary, **ALR**

SEATTLE

Adams, Frederick W.....
....Roosevelt Clinic, 1114 Boylston av., **OALR**
Bell, Will Otto....Medical-Dental bldg., **OP**
Bentley, Frederick....Cobb bldg., **OP**
Chase, E. Frank....Cobb bldg., **OALR**
Cohen, Bernard M., Lt. (M.C.), U.S.N., Section Base,
Cold Bay, Alaska, c/o Fleet Post Office, Pier 41, **ALR**
Dowling, J. Thomas....Terry av., **OALR**
Goss, Harold Leroy....4502 E. 33rd, **OP**
Hoffman, W. F.....1317 Marion, **OP**
Jensen, Carl Dana....Medical and Dental bldg., **OP**
Klotz, Wm. F., Lt. Col. (M.C.),
186 Station Hospital, A.P.O. No. 948, **ALR**
McCoy, L. L.....1317 Marion, **OP**
Miller, Alvin R.....Lt. (M.C.), Fourth and Pike bldg., **ALR**
Peden, Barton Edward....Stimson bldg., **ALR**
Shannon, C. W.....New 4th and Pike bldg., **OP**
Stillson, H.....Seabord bldg., **OALR**
Tolan, John F.....1115 Terry, **ALR**
von Zelinski, W. F., Col. (M.C.), Headquarters
N.W. Service Command, A.P.O. 702, c/o Postmaster, **OP**
Weber, Julius Albert....Stimson bldg., **ALR**

SPOKANE

Epstein, Sidney....Lt. (M.C.), Baxter General Hospital, **OP**
Mullen, C. J.....Maj. (M.C.), East 827 Overbluff, **OP**
Parsell, Louis A.....Paulsen Med. and Dental bldg., **ALR**
Rott, O. M.....1715 West Point road, **OALR**
Smith, Carroll L.....Paulsen Med. and Dental bldg., **OALR**
Veasey, C. A., Sr.....Paulsen Med. and Dental bldg., **OP**
Veasey, C. A., Jr.....Paulsen Med. and Dental bldg., **OALR**

TACOMA

Howe, A. W.....Medical Arts bldg., **OALR**
Ludwig, William Henry....Medical Arts bldg., **ALR**
Lundvick, Cyril V.....Medical Arts bldg., **OP**
Paul, William C.....Medical Arts bldg., **ALR**

VANCOUVER

Dunnavan, Floyd Leighton....Arts bldg., **OP**
Sugar, H. Saul..Capt. (M.C.), Barnes General Hospital, **OP**

WALLA WALLA

Bailey, Paul....Maj. (M.C.), McCaw General Hospital, **ALR**
Gowen, George Robert....Baker bldg., **ALR**
Stevens, Ralph W.....Baker bldg., **OP**

WEST VIRGINIA

BLUEFIELD
St. Clair, C. T. ..... Cor. Bland and Ramsey, ALR

CHARLESTON
Chandler, A. C. ..... Capt. (M.C.), 1562 Virginia st., OP
Holcombe, Virgil Eugene ..... Medical Arts bldg., ALR
Seletz, Abraham ..... Medical Arts bldg., ALR

CHARLES TOWN
Barlow, Roy A. ..... Box 333, ALR

CLARKSBURG
Thomas, Harry Vernon ..... Empire Bank bldg., OP
Weinberg, Herman C. ..... Lt. (M.C.), U. S. Army Induction Board No. 10, OP

ELKINS
Goodwin, Thomas M. ..... Golden Clinic, ALR

FAIRMONT
Johnson, H. R. ..... 320 Jefferson, OALR
Maxwell, Joseph Sprague ..... Professional bldg., OP
Traugh, George H. ..... 309 Cleveland av., ALR

HUNTINGTON
Beckner, William F. ..... 1050 Fifth av., ALR
Moore, J. Hallock ..... 1050 Fifth av., ALR
Moore, T. W. ..... 1050 Fifth av., OALR

MONTGOMERY
McGehee, Melvin Wood ..... Laird Memorial Clinic, ALR

MORGANTOWN
Wylie, C. B. ..... 293 High, OALR

PARKERSBURG
England, Welch ..... 1009 Market, ALR
Gilmore, Milton A. ..... Box 172, OP
Hartman, Eugene C. ..... 610½ Market, OALR

WELCH
Armentrout, Lewis Hamilton, Jr. ..... Grace Hospital, ALR
Schiefelbein, Harry T. ..... Stevens Clinic Hospital, OP
Vermillion, James Stephen ..... Capt. (M.C.), First Nat'l Bank bldg., ALR

WHEELING
Fawcett, Ivan ..... 75 Twelfth st., OALR
Jones, Edmund Lloyd ..... Wheeling Clinic, OALR
Tomassene, R. A. ..... 1144 Market, OALR
Zubak, Matthew F. ..... Citizens Mutual Trust bldg., OALR

WHITE SULPHUR SPRINGS
Croll, Leo J. ..... Capt. (M.C.), Ashford General Hospital, ALR
Hert, C. B. ..... Maj. (M.C.), Ashford General Hospital, ALR
Masters, John M. ..... Maj. (M.C.), Ashford General Hospital, OP

WISCONSIN

APPLETON
Brooks, E. H. ..... Zuelke bldg., OALR
Recter, A. E. ..... Zuelke bldg., OALR

ASHLAND
Grigsby, Roll O. ..... 100 E. Second, ALR

BELOIT
Shearer, Harry Arthur ..... 405 E. Grand av., ALR

EAU CLAIRE
Payne, A. L. ..... 119 S. Barstow, OALR
Russell, Sidney Benteen ..... Midelfart Clinic, OP
Tanner, James Wallace ..... 131 S. Barstow, ALR

FOND DU LAC
Folsom, W. H. ..... 11 N Main, OALR

GREEN BAY
Comee, W. C. ..... Northern bldg., OALR
Ford, William W. ..... 109 East Walnut, ALR
Nadeau, E. G. ..... Bellin bldg., OALR
Troup, Wilson J. ..... 306 Cherry, ALR

JANESVILLE
Pember, Aubrey H. ..... 500 W. Milwaukee, OALR

KENOSHA
Ashley, T. W. ..... 5711 Eighth av., OALR

LA CROSSE
Douglas, Frederick Alexander ..... State Bank bldg., OALR
Smith, D. S. ..... State Bank bldg., OALR

MADISON
Dayis, F. A. ..... 224 W. Washington av., OP
Dimond, Waldo B. ..... 16 N. Carroll, ALR
Duehr, Peter Alexander ..... 224 W. Washington av., OP
Lynch, Joseph S. ..... Capt. (M.C.), A.A.F.T.T.S., Truax Field, OP
Harned, John W. ..... Maj. (M.C.), A.A.F.T.S., ALR
Neff, Eugene E. ..... 224 W. Washington av., OP
Nesbit, Mark Edwin ..... 1 S. Pinckney, OP
Nesbit, Wellwood ..... 1 S. Pinckney, ALR
Schubert, Clarence K. ..... 1 S. Pinckney, OP
Smith, R. B. ..... 224 W. Washington av., ALR

MANITOWOC
Andrews, M. P. ..... 826 S. 8th, OALR
Schneck, Nathan ..... 815½ S. 8th, ALR

MARINETTE
May, J. V. ..... 1703 Main, OALR

MARSHFIELD
Copps, Lyman Alden ..... Marshfield Clinic, OALR

MILWAUKEE
Bach Edwin Clarence, ... 324 E. Wisconsin av., ALR
Blankstein, Samuel S. ..... 600 Wisconsin av., OP
Brook, Jeffrey J. ..... 231 W. Wisconsin av., ALR
Carl, Evan Elsworth ..... 606 W. Wisconsin av., OP
Chapman, Vernon A. ..... 324 E. Wisconsin av., OALR
Fox, Meyer S. ..... 606 W. Wisconsin av., ALR
Gordon, J. S. ..... Bankers bldg., OALR
Grove, W. E. ..... 334 E. Wisconsin av., OALR
Haessler, F. Herbert ..... Wells bldg., OP
Heeb, Harry J. ..... 212 W. Wisconsin av , OALR
Higgins, S. G. ..... 411 E. Mason, OALR
Hitz, H. B. ..... 411 E. Mason, ALR
Judge, Thomas Ambrose ..... 735 N. Water, ALR
Light, George ..... 709 N. 11th, ALR
McCormick, Thomas F. ..... Wells bldg., ALR
Martin, Hilmar George ..... 231 W. Wisconsin av., OP
Moisberry, Jasper Merl ..... 425 E. Wisconsin av., OP
Schmidt, Herbert G. ..... 411 E. Mason, OALR
Schoofs, Orlando P. ..... 324 E. Wisconsin av., ALR
Sproule, Ralph P. ..... 208 E. Wisconsin av., OALR
Tolan, Thomas L. ..... Wells bldg., ALR
Treskow, Frank G. ..... 411 E. Mason, ALR
Waldeck, Edward A. ..... 238 W. Wisconsin av., OALR
Warner, Raymond C. ..... 324 E. Wisconsin av., OP

MONROE
Kundert, Fred W. ..... The Monroe Clinic, ALR

OSHKOSH
Ebert, Reinhold O. ..... 104 Main, OP

WISCONSIN — Continued

RACINE
Beeson, Henry B. — 423 Main, ALR

SHEBOYGAN
Neumann, William H. — Sheboygan Clinic, OALR

STEVENS POINT
Dunn, Austin Gibbons — 111½ Strongs av., ALR

SUPERIOR
Myers, Elmer A. — Board of Trade bldg., OALR

WAUSAU
Trumbo, James Keeley — 526 Third, ALR
Wilson, Otis M. — 502 3rd st., OP

WHITEHALL
Simons, Neal S. — MacCornack Clinic, ALR

WYOMING

CASPER
James, George Russell — Wyoming Nat'l Bank bldg., OP

CHEYENNE
Newman, Erwin W. — 1606 Capitol av., OP

FORT F. E. WARREN
Wilson, Wm. H. — Capt. (M.C.), Station Hospital, ALR

SHERIDAN
Veach, Oscar Lloyd — Whitney Trust bldg., OP

CANADA

BRITISH COLUMBIA

VANCOUVER
Boucher, R. B. — Medical-Dental bldg., OALR
Brown, W. Harold — Birks bldg., OALR

VICTORIA
Keys, Morton J. — Pemberton bldg., OALR

MANITOBA

BRANDON
McDiarmid H. Oliver — 35 Clement block, OALR

WINNIPEG
Macneil, Frank A. — Medical Arts bldg., ALR
Ramsey, Robert M. — Flight Lt., R.C.A.F., 320 Yale av., OP

NEW BRUNSWICK

ST. JOHN
Chipman, L. DeV. — 42 Coburg, OALR
Hayes, Robert T. — 42 Coburg, OALR

NOVA SCOTIA

HALIFAX
Schwartz, Hugh William — 163 S. Park, ALR

ONTARIO

CAMP BORDEN
Biehn, J. T. — Military Hospital, OALR
Henry, G. A. — Capt., R.C.A.M.C., Military Hospital, ALR

HAMILTON
Arrell, W. — 317 Main st. E., OALR
Macfarlane, P. B. — Medical Arts bldg., OALR
Rabinowitz, Paul — Mountain Sanitarium, ALR

LONDON
McFarlane, Donald C. — 380 Queens av., ALR

OTTAWA
Fraser, W. G. — Medical Arts bldg., OALR
Smart, Robert Edwin — Medical Arts bldg., OP

PETERBOROUGH
Buchanan, Norman D. — 129 Murray, OALR

TORONTO
Alexander, Seeyman L. — Medical Arts bldg., ALR
Atkins, John — 345 Bloor st. W., ALR
Barnes, Lancelot — 170 St. George, OP
Bochner, Maxwell K. — 329 Bloor st. W., OP
Burnham, Howard Hampdon — Medical Arts bldg., ALR
Campbell, A. A. — 86 Bloor st. W., ALR
Davies, T. A. — 578 Sherbourne, OALR
Goldsmith, Perry G. — Medical Arts bldg., ALR
Gollmon, Joseph — 425 Bloor st. W., ALR
Hill, C. E. — 160 Bloor st. W., OP
Ireland, Percy E. — 68 High Park av., ALR
Luke, William R. F. — Medical Arts bldg., OP
McCart, Howard W. D., R.C.A.M.C., Maj. 15th General Hospital, Canadian Army Overseas, ALR
McCulloch, Robert J. P. — Medical Arts bldg., OALR
McEachern, Margaret Aileen — Medical Arts bldg., ALR
McGregor, Gregor William — 170 St. George, ALR
McLeod, Angus M. — Medical Arts bldg., ALR
Morgan, Alonzo Lloyd — 170 St. George, OP
Nugent, M. W. — Flight Lt., R.C.A.F., 37 Strathearn rd. W., OP
Pentecost, R. S. — 338 Bloor st. E., OALR
Risdon, E. F. — Medical Arts bldg., ALR
Robert, Joseph X. — 170 St. George, ALR
Smith, I. Ruebert — 116 College st., ALR
Snell, George C. — Medical Arts bldg., ALR
Stark, W. Berkeley — Medical Arts bldg., ALR
Sullivan, Joseph A. — 170 St. George, ALR
Vaile, Ivan R. — 384 Bloor st. W., ALR
Veitch, Ambert H. — 170 St. George, ALR
Wishart, David Edmund Staunton — Medical Arts bldg., ALR
Wright, Walter Walker — Medical Arts bldg., OP

WELLAND
Briant, Thomas Ernest — 28 East Main, ALR

QUEBEC

MONTREAL
Ballon, D. H. — 1471 Crescent, ALR
Freedman, Abraham O. — 1396 St. Catherine st. W., ALR
Hodge, George E. — 1414 Drummond, ALR
Hutchison, Keith O. — 1509 Sherbrooke st. W., ALR
Johnston, Kenneth B. — 1509 Sherbrooke st. W., OP
Lapointe, Jean — 196 Willowdale, ALR
Lundon, A. E. — Col., 1463 Bishop st., Apt. C-3, ALR
McAuley, Albert G. — 1301 Dorchester st. W., OP
McNally, William James — Linton Apartments, ALR
MacMillan, J. A. — 1410 Stanley, OP
Ramsey, G. Stuart — 1496 Mountain, OP
Rogers, J. T. — Medical Arts bldg., ALR
Rosenbaum J. — 1396 St. Catherine st. W., OP
Tremble, George E. — 1930 Sherbrooke st. W., ALR
Wright, R. Percy — 6065 Sherbrooke st. W., ALR

BRITISH WEST INDIES

TRINIDAD
Weiss, J. Allan, Lt. Comdr. (M.C.), U. S. Naval Air Station Dispensary, ALR

PUERTO RICO

SAN JUAN

Fernandez, Luis J...Instituto Oftalmico de Puerto Rico, **OP**
Fernandez, Richardo F......No. 15 Ponce de Leon av., **OP**
Font, Juan H. P. O. Box 5363,
Puerta de Tierra Station, Medical Arts bldg., **ALR**

UTUADO

Carrasquillo, Honorio Fernando.....
.....P. O. Box 128, Puerto Rico, **OP**

AFRICA

NATAL

DURBAN

Johnson, Lindsay.....4 Evans road, **OP**

CENTRAL AMERICA

CANAL ZONE
COCO SOLO

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